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SKIN GRAFTING IN SURGERY FOR CARCINOMA OF THE BREAST

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For many years skin grafting has been a method of closing defects on the chest wall after mastectomy for carcinoma. When Halsted described his radical mastectomy in 1894, he recommended skin grafting for the repair of both the thoracic and axillary defects in those cases where wound closure by direct approximation of the skin edges was not possible.

Where adequate removal of the skin, therefore, leaves a defect which cannot safely be closed by simple undermining and approximation, closure by local flaps or by importing tissue from elsewhere in the form of free grafts or pedicles is required. An important aspect of this matter is the need for its recognition pre-operatively in cases to which it applies. Local flaps, which are undertaken *ad hoc* and haphazardly on the table, are nearly always bad flaps and may only cause additional necrosis and scarring.

On the whole, Thiersch grafting is the safest method of closing large defects, and it causes no additional scarring. Local flaps are in any case contraindicated in tissues which have been heavily irradiated and which show resultant dermatitis or fibrosis. Here skin grafting is the method of choice.

Although skin grafting as a routine counterpart of the Halsted radical mastectomy has been abandoned by most surgeons, it is still required in all those cases where, after mastectomy, the defect cannot be closed by approximation of the skin edges without undue tension or by safe local flaps. This is particularly true for carcinomas of the medial quadrant of the breast. In small breasts, with the removal of skin of an adequate diameter, skin grafting may also be found necessary.

Thiersch grafting may be required (in addition) where mastectomy, either simple or radical, or whether by intention curative or palliative, is carried out for group III carcinomas involving the skin, especially where there is ulceration of the overlying skin. Wide excision and Thiersch grafting may be the treatment of choice.

Figs. 1 and 2 illustrate such a case of a stage III fungating carcinoma. This 51-year-old woman first had a full course of deep X-ray therapy to the breast and regional lymphatics. Subsequently a bilateral oophorectomy was performed. All this produced only temporary improvement, and when seen in the department of plastic surgery at St. Bartholomew's Hospital, London, there was a raised, ulcerating mass to the right of the nipple, 2 inches in diameter and oozing blood and serous fluid (Fig. 1). The whole breast was enlarged and indurated, a mass of infiltrated tissue spreading below the right axilla. There was no palpable lymphadenopathy. Chest X-ray was normal.

A total mastectomy was performed with wide skin excision and axillary clearance. The skin edges were approximated in the medial inferior quadrant of the right chest wall and the right axillary region. A large oval defect, measuring 5×7 inches, was covered with a split-skin graft taken from the thigh.

Fig. 2 is a postoperative photograph at 8 months. The graft was stable and the whole area completely satisfactory.

Thiersch grafting is furthermore often required after mastectomy for carcinoma of the male breast. In sarcoma of the breast it is generally unavoidable owing to the extensive skin adhesion and the amount of skin that has to be removed.

Lastly, I feel that excision and grafting also has a place in the treatment of local recurrence of carcinoma after surgery. It is a moot point whether at mastectomy very wide excision and closure of the defect with a Thiersch graft (as originally recommended by Halsted), or limited excision of skin with wide undermining (as recommended by Handley), is the better method to avoid local recurrence. A high incidence of local recurrence (approximately 20% of patients operated upon) has been reported in most large series regardless of which of the above methods was followed. The advent of roentgen therapy and thorough pre- and postoperative treatment of the operative field have also failed to reduce the incidence of local recurrence.

This recurrence after proper radical operation may result from the physical impossibility of removing all cancerous tissue in the internal mammary and intercostal areas. At operation there may already be undetectable inoperable extension of the cancer. Here the local recurrence is the result of retrograde extension (permeation and embolization) along residual intercostal and internal mammary lymphatics. In these cases the recurrence is often widespread and diffuse, extending from between the ribs and involving the deeper chest wall. Further surgery is seldom possible.

In the second place, local recurrence may arise from malignant inoculation at the time of operation. The recurrence follows a transplantation of cancer cells cut into in the course of the operation. These recurrences commonly occur along the mastectomy scar. Here there is a place for wide re-excision and Thiersch grafting, since these patients often do not respond to further radiotherapy and/or endocrine therapy. Fig. 3 is an example of a case of this nature.

This 69-year-old woman had a total mastectomy and axillary clearance for carcinoma. Soon after the operation she developed an ulcer in the operation scar. Biopsy confirmed the recurrence of an undifferentiated spheroidal-cell carcinoma of the breast. This steadily growing mass, over the next months, stubbornly resisted both radiotherapy and later endocrine treatment.

When referred to the department of plastic surgery at

St. Bartholomew's Hospital, London, she was seen to have a fungating mass, 3 inches in diameter, in the mastectomy scar (Fig. 3). There was no palpable lymphadenopathy, and the chest and rib X-rays showed no abnormality.

At operation the mass was widely excised and removed down to underlying ribs of which it seemed free; histological section later confirmed this. The defect on the chest wall, measuring 4×5 inches, was closed with a thick, split-skin graft from the thigh.

Six months after the operation the patient was symptom-free and well. The graft was stable and there was no further evidence of recurrence (Fig. 4).

This is of course no proof that the local condition has been cured, or that there is no distant extension of the carcinoma. For 6 months the patient has, however, been free of a foul-smelling, malignant ulcer which, over a period of many months, had not responded to any other form of treatment.

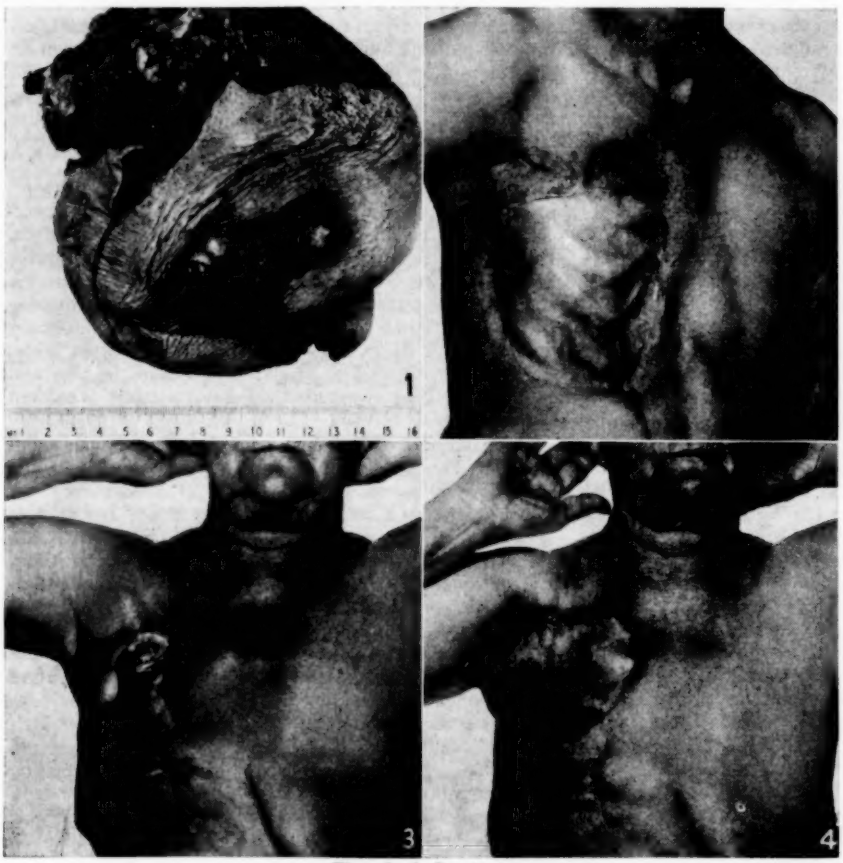
This in itself is sufficient justification to try excision and grafting in these otherwise hopeless cases.

The skin grafting itself presents no special problems. The grafts almost invariably 'take' well. There are, however, a few points to remember. The skin edges, which have usually been undermined during mastectomy, may be brought in to diminish the size of the defect. It is important to tack these flap edges down well to the chest wall with interrupted silk sutures, before applying the graft to the remaining defect. This prevents leaking of blood under the grafts, and serious oozing originating under the flaps. Drains should be inserted under the flaps with their exits dependant and at some distance from the graft. After clearance of the axilla, a separate drain should be left *in situ* there.

When the operator is satisfied that the bed for the graft is absolutely dry, a thick split-skin graft is taken from the thigh. It is important to change gowns and gloves after removing the tumour before taking the skin graft, since carcinoma cells can be transplanted to the donor site. The development of adenocarcinoma in the donor site has been described.

The graft is applied, if possible, in a single large sheet. It is advisable to puncture the graft in several places to allow escape of serum. It is then sutured in place, leaving some of the sutures long. These are used to tie a pad of flavine wool or plastic sponge, on a single layer of tulle gras, into place over the graft. This provides accurate local pressure. The chest is then covered in a liberal dressing of cotton wool, taking special care to pack the axilla well in order to prevent any collection of blood or serum there. The usual large spica dressing may then be applied.

Using this technique it is possible to change the outer dressings and remove the drains after 48 hours without disturbing the graft. The tie-over graft dressing is removed on the seventh postoperative day.



Figs. 1 - 4. See text.

SUMMARY

If, after radical mastectomy for breast carcinoma, skin closure by simple undermining and approximation of the wound edges is impossible, Thiersch grafting is indicated. The technique for this is described. It is stressed that there is a place for wide excision and grafting in the treatment of local recurrence on the chest wall after mastectomy.

I wish to thank Mr. P. H. Jayes, Head of the Plastic Surgery Unit at East Grinstead and consultant plastic surgeon to St. Bartholomew's Hospital, London, for entrusting the cases described in this article to my care.

EDITORIAL : VAN DIE REDAKSIE

MEDICAL HISTORY

Two reports which recently appeared in this *Journal* have given us particular pleasure. The first announced the establishment of a Museum of the History of Medicine by the Medical Graduates Association of the University of the Witwatersrand.[1] The second gave an account of the first meeting of a Medical History Club at the University of Cape Town.[2] The development of an interest in one's historical heritage is a sure sign of maturity, and it is fitting that our two senior medical schools should be demonstrating their coming of age in this fashion.

Until now, the study of medical history has not avidly been pursued in Southern Africa. There have been, however, some notable individual exceptions. A former Editor of this *Journal*, Dr. C. L. Leipoldt, often wrote on medical history topics; Dr. E. H. Burrows, in his *History of Medicine in South Africa*, has provided a valuable introduction to the early medical history of this country; and Dr. M. Gelfand is soon to publish the last volume of a trilogy in which the medical history of Nyasaland and the Rhodesias is carefully recorded. There is little doubt that the medical museum in Johannesburg and the club in Cape Town will stimulate a much wider interest in this fascinating subject.

It has often been said that medical history is the subject to which elderly practitioners gratefully turn when they no longer dare to contemplate the realities of the present or the future. This is no longer true. The great increase in the study of medical history which is occurring in this country and throughout the world has been stimulated by the increasing participation of many young men.

Those who are closest to the advancing frontiers of medical science now realize that the innumerable new discoveries which are being proclaimed every day can only be assessed properly when studied in their historical perspective. To take an extreme example: those who are familiar with the long and chequered history of 'cancer cures' will be less inclined to accept the claims made for modern panaceas without the evidence of very thorough and protracted trials. When he is searching for new approaches to the solution of an old problem, the young research worker, who studies the historical development of the ideas about his subject, will find that, as he follows this current of ideas from the past to the present, his own stream of thought will gain increased momentum for its projection into the future; the more carefully he delves back into the past, the more clearly will he see the path to future discovery.

These, we believe, are the most important benefits which are to be derived from a study of medical history. There are others which we may mention. It is right and proper that due regard and respect be paid to the great medical men of the past and that we do not forget the tremendous discoveries on which modern medicine is based. The anniversaries of the great politicians and of their wars of destruction are enthusiastically celebrated in every country, and they are honoured annually by national holidays and festivals. There is at least as much reason for us to commemorate the great benefactors of mankind and their victories over disease. The student of the history of medicine is brought into close contact with non-medical disciplines, and his links with general history, classical culture, art, music, and literature are strengthened — in fact medical history is the most effective means that we have of bridging the gap between what Sir Charles Snow has called the 'two cultures' — the arts and the sciences. In this way medical men will be able to demonstrate the importance of health and disease in shaping the destiny of individuals and of nations; in turn, they will learn how much medical progress is dependent on the progress of humanism and science in general.

Finally there are some less profound, but not less important, virtues to be found in the study of medical history. It can provide an absorbing hobby and useful relaxation for those who require it. It provides medical teachers with interesting material with which to brighten a potentially dull lecture and, for didactic purposes, the 'historical approach' is often a convenient method of explaining a complicated subject. For those doctors' wives who would like to take a more active interest in their husbands' profession, there are many facets of medical history which can with advantage be studied by husband-wife teams. In the past many important contributions to medical history have been made by the combined efforts of a doctor and his wife. We anticipate that for these and many other reasons our new medical history organizations will be welcomed warmly and that they will prosper and flourish.

1. Medical Graduates Association (1961): S. Afr. Med. J., **35**, 527.
2. UCT Medical History Club (1961): *Ibid.*, **35**, 761.

DIE LIG VAN VERRE DAE

Dit lê in die aard van die tyd waarin ons leef dat ons kennis al meer in nouer vakke verloop en al meer en meer hoogs gespesialiseerd word. Op 'n skaal wat heeltemal ongeëwenaard is, is tallose navorsers orals oor die wêreld besig om na die diepte van dinge te peil — 'n bedrywigheid wat deur 'n duisend en een moderne tegniese en wetenskaplike hulpmiddels moontlik gemaak en aangemoedig word. Die totale som van ons kennis vermeerder waarskynlik *elke dag* meer as wat dit 'n honderd jaar gelede *per jaar* of selfs *per dekade* vermeerder het.

Hierdie toestand van sake het dus onvermydelik geword, maar dit hoef nie noodwendig 'n onvermengde euwel te

wees nie, mits ons alles in ons vermoë doen om ons blik en uitsig nie te laat beperk nie en ons perspektief nie te laat belemmer nie. Om hierdie rede is dit dus goed en verblydend dat daar tekens is van 'n hernieude belangstelling in mediese geskiedenis en in die breër filosofiese, wetenskaplike, artistieke, kulturele, en algemeen-menslike agtergrond van die mediese kuns en wetenskap.

Geskrifte[1-4] wat afkomstig is van lede van die doserende personeel van die Universiteite van Pretoria, Stellenbosch, en Durban, het reeds al getoon dat die behoefte aan die navraag na die herkoms van ons kennis en gebruike 'n bewuste geestesinhoud geword het. En onlangs het hierdie neiging 'n nuwe impetus gekry aan die oudste twee van ons mediese skole, deur die stigting van 'n mediese museum[5] aan die Universiteit van die Witwatersrand, onder die geesdriftige leiding van dr. Cyril Adler, en die stigting van die Klub vir Mediese Geskiedenis[6] aan die Universiteit van Kaapstad, onder die besielende leiding van dr. Hymie Gordon. Ons hoop en vertrou dat hierdie pogings allerweë aangemoedig sal word, omdat die studie van die geskiedenis van die medisyne een van die maniere is waarop ons ons perspektief kan verbreed.

Aan sommige van die ouere mediese skole op die Vasteland van Europa en in Amerika word die geskiedenis van die medisyne as vak gedoseer. Dit is 'n gewenste toestand van sake. Of dit deesdae nog prakties uitvoerbaar is, in die lig van die groot omvang van nuwe kennis wat maar steeds by die leerplan ingevoeg word, is twyfelagtig. Maar, dat daar iets daadwerkliks in hierdie verband gedoen moet word, val nie te betwyfel nie. Daarom is dit 'n goeie benadering om opsionele fasiliteite vir 'n studie van hierdie aard daar te stel — veral as dit gedoen kan word rondom die kern van 'n bestaande klub of museum.

Die wortels van ons mediese kennis en gebruike gaan ver terug in die verlede, en 'n studie van hierdie agtergrond kan baie daaraan doen om vir ons 'n insig te laat kry in die menslike persoonlikheid. Die mens mag vandag baie anders voorkom as vroeër, en hy mag leef teen 'n ongekende tempo en met die fasade van volkome volwassenheid. Tog is dit nie so nie. Die mens het in sy wesenlike aard baie dieselfde gebly, en onder die dop van tegniese vaardigheid skuil die basiese onsekerheid van almal nog altyd.

Om die lig van verre en vergange dae op ons huidige doen en late te laat skyn, kan 'n bevrugtende en behoudende uitwerking hê. Miskien sal dit juis goed wees om na te speur hoe eenders die wesenlike mens tog maar is, ten spyte van sy skynbare andersheid. Daarom is die hele gebied van die lewe en strewe van die mens dwarsdeur die historiese tydperk vir ons van belang.

Ons doel met die studie van die geskiedenis van die medisyne moet dus wees, nie net om ons eie prestasies op te hemel nie, maar eintlik om die mens as mens beter te leer ken en te verstaan. Dan sal ons moontlik kan uitvind waarom groter geeste as ons in die verlede gefaal het. En dan sal ons ook 'n insig kan kry in die gevaarpunte wat vir ons vandag as waarskuwings moet dien.

Om die studie van die geskiedenis van die medisyne werklik vrugbaar te maak, is dit nodig dat ons die agtergrond van die uitspraak van Emile Littré werklik leer ken en verstaan — 'n uitspraak wat meer as 'n honderd jaar gelede al gemaak is en nou nog net so waar is as toe: ‚as die mediese wetenskap nie tot die vlak van 'n blote meganiese professie moet daal nie, moet hy homself besig hou met 'n studie van sy geskiedenis'.

1. Snyman, H. W. (1960): S. Afr. T. Geneesk., **34**, 422.
2. Brede, H. D. (1961): *Ibid.*, **35**, 716.
3. Steytler, J. G. (1961): *Ibid.*, **35**, 760.
4. Gordon, I. (1960): *Ibid.*, **34**, 414.
5. Medical Graduates Association (1961): *Ibid.*, **35**, 527.
6. UCT Medical History Club (1961): *Ibid.*, **35**, 761.

ACUTE RENAL FAILURE — ITS DIAGNOSIS AND MANAGEMENT, INCLUDING THE USE OF THE TWIN-COIL ARTIFICIAL KIDNEY

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Acute renal failure may be defined as any abrupt deterioration in renal function resulting in azotaemia and disturbance of electrolyte metabolism, usually associated with oliguria (400 ml. per 24 hours or less*). This, however, is not invariable, since instances of sudden renal failure without oliguria are encountered. This is distinct from missed oliguric renal failure, where the oliguric phase has lasted only a few hours, the patient presenting in the diuretic phase.

Its causes are many and diverse. There is virtually no field of medicine or surgery in which this condition may not be encountered; hence all clinicians should be familiar with the basic principles in the diagnosis and management of acute renal failure. The prognosis of the condition has improved vastly with the introduction of the 'conservative régime',[1,2] and there can be little doubt concerning the additional beneficial results of haemodialysis. The use of the artificial kidney should no longer be regarded as a heroic measure in a moribund patient. Today the safety and the value of haemodialysis is accepted; it is now a recognized therapeutic procedure to be considered seriously in any patient with acute renal failure. The pendulum has swung (perhaps too far) from occasional use as a desperate measure to routine daily or prophylactic dialysis.[3,4]

In Groote Schuur Hospital 55 patients with acute renal failure have been treated during the period that the Kolff disposable-coil kidney has been in operation. The advisability of earlier dialysis has become apparent, and it is therefore of paramount importance that patients be referred to experienced specialized renal-insufficiency units as soon as possible. This paper records 2 years' experience in the diagnosis and management of acute renal failure with particular reference to the use of the artificial kidney.

* This is an arbitrary level (see later).

DIAGNOSIS

Acute renal failure is a clinical syndrome like hepatic or pulmonary failure. The majority of cases of acute renal failure are attributable to acute tubular necrosis. The variety of associated conditions and other relevant clinical details of the 55 patients are set out in Table I.

While efficient management of acute oliguric renal failure is dependent on early recognition, there are certain difficulties in the diagnosis.

TABLE I. ACUTE RENAL FAILURE—CLINICAL DETAILS OF 55 CASES

No.	*Race*	*Sex*	*Age (years)*	*Diagnosis*	*Remarks*	*Duration of oliguria (days)*	*1-l. day **	*Day of dialysis*	*Peak blood urea (mg. per 100 ml.)*	*Peak serum potassium (mEq./l.)*	*Outcome ***
Obstetric:											
1	B	F	40	Accidental haemorrhage (34 weeks). Afibrinogenaemia	Persistent bleeding. 10 pints blood. Hypokalaemia during diuretic phase	19	23	11, 17	452	7·5	R
2	C	F	46	Concealed accidental haemorrhage (32 weeks)	Caesarean section—oligaemic shock. 6 pints blood	9	10	8	287	7·1	R
3	C	F	21	Concealed accidental haemorrhage (34 weeks)	Hypotension for 9 hours. 2 fits. 8 pints blood	17	22	12, 19	346	8·4	R
4	C	F	30	Concealed accidental haemorrhage (34 weeks)	Eclamptic fits. 4 pints blood	13	15	5	295	7·1	R
5	B	F	42	Concealed accidental haemorrhage (32 weeks)	4 pints blood. High potassium from inadvertent potassium administration countered with 'CTS-3 resin'. Late urinary-tract infection	11	13	8	425	6·8	R
6	B	F	50	Spontaneous abortion (? 20 weeks)	Rapid recovery. 3 pints blood	4	6	—	184	6·0	R
7	C	F	31	Infected abortion (10 weeks)	*Cl. welchii* infection. Pelvic peritonitis. Severe parotitis. 3 pints blood	15	20	10	560	10·0	R
8	C	F	36	Incomplete abortion (12 weeks)	Retained placenta. Septicaemia and prolonged oliguria	29	37	16	475	6·9	R
9	E	F	36	Infected abortion (12 weeks)	Septicaemia and peritonitis. 2 pints blood. Called on 6th day to jaundiced, overhydrated, moribund, patient	7	—	7	600	5·3	D— 7
10	C	F	25	Infected abortion (12 weeks)	Perforated vaginal vault with severe diarrhoea. Pelvic abscess. Septicaemia and acute endocarditis. Gross jaundice. Serum bilirubin 57 mg. per 100 ml.	16	19	11	310	5·2	D—19
11	B	F	26	Infected abortion (12 weeks)	Pelvic peritonitis. Intravascular haemolysis. Serum bilirubin 2·2 mg. per 100 ml. 2 pints blood	15	17	10	360	5·4	R
12	C	F	39	Infected abortion (12 weeks)	*Cl. welchii* infection. Serum bilirubin 1·5 mg. per 100 ml. 2 pints blood	13	17	—	422	6·0	R
13	C	F	19	Infected abortion (24 weeks)	*Cl. welchii* infection. 3 pints blood	12	17	—	560	6·9	R
14	B	F	35	Incomplete abortion (20 weeks)	Prolonged diuretic phase—35 days. Severe persistent diarrhoea since 11th day	4	5	—	510	5·5	R
15	B	F	23	Infected abortion (12 weeks)	Staphylococcal septicaemia. Pulmonary abscess. 6 pints blood. Recovered from renal failure. Normal blood urea for 12 days	5	6	—	242	5·5	D—22
16	C	F	20	Infected abortion (12 weeks)	Potassium remained low throughout. Persistent vomiting	15	18	—	321	4·8	R
17	C	F	20	Infected abortion (16 weeks)	*Cl. welchii* infection with intravascular haemolysis—bacteriogenic pan-agglutin present. 3 pints blood	14	18	10	380	4·9	R
18	C	F	34	Infected abortion (12 weeks)	Pelvic peritonitis	25	27	6, 16	410	7·4	R
19	C	F	40	Infected abortion (14 weeks)	Gross herpes febrilis. Peritonitis. Intravascular haemolysis ++ before transfusion. Serum bilirubin 26·5 mg. per 100 ml. 2 pints blood	11	14	7, 13	555	7·4	R
20	C	F	25	Incomplete abortion (8 weeks)	Mild jaundice. 3 pints blood	18	23	—	520	4·9	R
21	C	F	28	Infected abortion (8 weeks)	Herpes febrilis. *Cl. welchii* infection. 4 pints blood	5	7	—	280	3·8	R
22	C	F	25	Infected abortion (12 weeks)	Herpes febrilis. Pelvic peritonitis. Intravascular haemolysis ++. Serum bilirubin 20·8 mg. per 100 ml.	8	13	7, 10	441	5·6	R
23	C	F	24	Post-traumatic abortion (18 weeks)	Kicked in abdomen. Postabortal haemorrhage	22	27	18	239	4·4	R
24	B	F	32	Intra-uterine death—premature induction. Postpartum haemorrhage	Overhydration. Extreme oliguria. 3 pints of blood. PM: Fibrinoid necrosis of interlobular arteries. 'Failed reflow' kidney and mild proximal-tubule necrosis	17	—	9	420	6·9	D—17
25	B	F	40	Postpartum eclampsia	Collapsed suddenly. PM: Renal tubular regeneration. Eclamptic lesions in liver	4	—	—	148	6·0	D— 5
Nephrotoxic:											
26	C	M	27	Mercuric biniodide poisoning	Misdiagnosed elsewhere as having an 'acute abdomen'—laparotomy performed	10	12	10	450	5·8	R
27	C	M	29	Ol. chenopodium poisoning	Hepatorenal failure. Perforation and peritonitis	5	7	—	392	4·0	R
Miscellaneous:											
28	B	F	67	Extensive burns	? Transfusion reaction. Fulminating diarrhoea since day 7—? non-specific enterocolitis	9	—	—	386	6·2	D—19
29	E	F	58	Hepatorenal failure of unknown aetiology	Slow diuresis. ? Nephrotoxic agent. Severe bronchopneumonia	10	17	10	392	5·7	R
30	C	F	68	Mesenteric thrombosis	Infarction of bowel. Chronic pyelonephritis	5	—	—	306	4·7	D— 5
31	E	M	35	Staphylococcal septicaemia	Acute pyelonephritis and osteomyelitis of ribs	6	7	5	315	6·1	R

No.	Race	Sex	Age (years)	Diagnosis	Remarks	Duration of oliguria (days)	1-l. day*	Day of dialysis	Peak blood urea (mg. per 100 ml.)	Peak serum potassium (mEq./l.)	Outcome**
32	E	F	59	? Periureteritis fibrosa	Intermittent oliguria lasting 2 or 3 days relieved by catheterization	—	—	—	317	7·6	R
33	B	M	35	Cerebral malaria	No true oliguric phase observed. No haemoglobinuria	**	9	—	378	5·2	R
34	E	M	50	Protocoproporphyria	Profound hypotension from peripheral circulatory failure—respiratory paralysis	—	—	12	269	6·2	R
35	E	M	44	Acute beriberi	Total anuria for 6 days. Bilateral renal-cortical necrosis. Infarction of papillary muscles of heart	9	—	7	510	5·7	D—9
Postoperative:											
36	C	M	75	Prostatectomy	Severe postoperative haemorrhage and pyaemia. Gross overhydration. PM: Acute pyelonephritis and acute tubular necrosis	9	—	8	400	6·4	D—9
37	E	M	67	Prostatectomy	Urinary infection treated with kanamycin	**	**	—	315	4·6	R
38	E	F	60	Laparotomy (hepatic haemangioma)	Severe haemorrhage from liver haemangioma	4	5	—	305	5·9	R
39	E	F	60	Laparotomy (melaena)	Cerebral haemorrhage with hemiplegia on day 8	8	—	—	204	4·9	D—8
40	C	F	28	Laparotomy (gastric ulcer)	Postoperative peritonitis	4	8	4	248	8·3	R
41	E	M	65	Aneurysmectomy	Popliteal embolism with gangrene. Pulmonary embolism	7	—	5	269	7·2	D—7
42	E	M	76	Aortectomy and graft	Postoperative cerebrovascular accident and myocardial infarction	10	—	8	581	6·8	D—10
43	E	M	21	Excision of aortic coarctation	Hypotension during operation	9	13	—	650	6·9	R
44	E	M	27	VSD repair (hypothermia)	Postoperative haemorrhage	4	15	—	437	4·4	R
45	E	M	28	VSD repair (hypothermia)	Cardiac arrest. Myocardial infarction. Pulmonary embolism. PM: Mild tubular necrosis	4	—	—	174	6·6	D—4
46	C	F	68	Hysterectomy (uterine polyp)	Known diabetic	4	8	—	133	5·4	R
47	E	M	65	Colectomy (polyp of colon)	Pulmonary embolism	9	—	—	203	5·8	D—9
48	E	M	65	Choledochostomy	Cholelithiasis. Suppurative cholangitis treated with kanamycin. Died of infection after recovery from renal failure	11	15	21	340	4·6	D—28
Post-traumatic:											
49	C	M	29	Multiple injuries	Intercurrent influenza. PM: Crushed chest, fracture of femur, acute tubular necrosis	10	—	6	471	6·9	D—10
50	B	M	45	Multiple injuries	Complete anuria from day 2. PM: Renal cortical necrosis	9	—	8	426	5·6	D—9
51	B	M	30	Multiple injuries	2nd dialysis during diuretic phase stopped because of severe gastro-intestinal haemorrhage	8	12	7, 14	490	6·4	D—15
52	E	M	26	Multiple injuries	Complete anuria from day 3. Pulmonary embolism. PM: Renal cortical necrosis	6	—	—	280	6·6	D—6
53	B	M	35	Multiple injuries	Overhydrated and jaundiced when seen on the 4th day. Bronchopneumonia and anoxia preterminally	6	—	4	232	8·5	D—6
54	C	M	30	Multiple injuries	Severe head injury. ? Fat embolism	5	—	—	255	5·6	D—5
55	B	M	49	Multiple injuries	Severe oligaemic shock. 5 pints blood	3	5	—	326	5·6	R

B = Bantu, C = Coloured, E = European, M = male, F = female, PM = postmortem findings, VSD = ventricular septal defect.

Under 'remarks', the amount of blood transfused initially is recorded in pints.

* '1-l. day' = day on which urine volume reached 1 litre.

** R = recovery, D = death, followed by the day of death.

Trauma, shock, blood loss, salt and water depletion — these and many other pre-renal factors lead to oliguria and azotaemia. This condition, which is here termed acute renal circulatory insufficiency, may be difficult to differentiate from acute renal failure caused by acute tubular necrosis. Apart from clinical assessment of dehydration and the observation of alterations in the specific gravity (SG) of the serum, and in haemoglobin and haematocrit values, this distinction is made in the first instance by directing attention to the urine and not to the serum chemistry.

The Importance of Investigation of Urine

Careful urine collection is essential — inaccurate measurements, failure to chart urine, urine mixed with bowel action, wetting the bed, and blocked catheters, are all likely to lead to a false assessment. The definition of oliguria has been a contentious subject, and upper limits have been arbitrarily set at 300 ml.,[2] 400 ml.,[6] 500 ml.,[6,7] 760 ml.,[5] and even 1,000 ml.[9] We accept 400 ml. as the upper limit, since, in our experience, once the urine volume reaches this figure a progressive stepwise increase follows, so that the '1-litre day' is reached 4 - 5 days later. While the presence of proteinuria and abnormal formed elements in the urinary sediment point to a primary renal condition, the measurement of the SG and analysis of the composition of the urine is of real assistance in diagnosis.

The measurement of the SG of the small amounts of urine available is often difficult, and should be carried out by the doctor and not by the nursing staff. While a high SG (> 1018) favours a pre-renal cause, a low SG (usually about 1010) does not necessarily mean renal failure. Physiological oliguria can be demonstrated to occur with a low or a high urine concentration,[6] depending on solute and water intake. Oliguria with a low SG may be encountered when sodium and urea excretion is low, as in renal circulatory insufficiency, and, postoperatively, following pre-operative restriction of protein and salt intake. Nevertheless, SG measurement when properly done is useful, and while it is not as refined as the estimation of urine osmolarity, it has the advantage of simplicity.

Urine-urea concentration is especially useful when very small amounts of urine are available. It may be high in the first instance, but with the evolution of tubular necrosis the concentration falls towards plasma levels — urea concentration is usually below 1·5 G. per 100 ml. (510 - 1029 mg. per 100 ml. in this series). A blood-urea concentration above 100 mg.

per 100 ml. and a urine concentration under 2 G. per 100 ml. has been held to favour acute renal failure, provided chronic renal disease can be excluded.[10]

The electrolyte pattern of the urine is variable; sodium concentrations have ranged from 43 - 102 mEq./l., chloride from 19 - 64 and potassium from 16 - 50. In general, in acute tubular necrosis urine-sodium concentration is almost invariably 'high' (> 35 mEq./l.), but may be low in certain cases — commonly following burns,[11] sometimes after trauma[12] and transfusion reactions,[28] and after pre-operative restriction of salt. Thus, while a low sodium concentration may sometimes occur in acute tubular necrosis, in general a high urinary-sodium concentration and a low urea favour this diagnosis. On the other hand, a low sodium and a high urea concentration favour acute renal circulatory insufficiency. This is also the pattern seen in acute glomerulonephritis.

Having made the diagnosis of acute renal failure, questions of aetiology and reversibility must be considered.

AETIOLOGICAL CONSIDERATIONS

Acute renal failure rarely occurs without apparent cause and, indeed, there is frequently evidence of more than one precipitating factor.

Careful questioning is important (ingestion of drugs and poisons, attempted abortion, previous state of shock, etc.). An inadequate history is frequently the result of ignorance or evasiveness on the part of the patient and, sometimes, the uraemic mental state precludes questioning. Interrogation of the relatives or friends may assist, but enquiries may be fruitless, particularly in cases of criminal abortion. Over 70

TABLE II. CAUSES OF ACUTE RENAL FAILURE

Intrinsic Renal Failure

1. *Acute tubular necrosis:*
   - Group I: Obstetric; nephrotoxic; intravascular haemolysis; and miscellaneous, especially 'hepatorenal' syndrome and infections
   - Group II: Postoperative; and post-traumatic, including 'crush syndrome'
2. *Bilateral cortical necrosis*
3. *Vascular obstruction:*
   Bilateral renal emboli
   Bilateral thrombosis of renal artery and vein
4. *'Hypersensitivity states':*
   Acute diffuse glomerulonephritis
   Polyarteritis nodosa
   Systemic lupus erythematosus
   Other hypersensitivity states, e.g. to penicillin
5. *Acute pyelonephritis:*
   Renal medullary necrosis
6. *Acute renal failure superimposed on chronic renal disease*

Extrinsic Renal Failure

1. *Prerenal (acute circulatory insufficiency):*
   Hypotension
   Haemorrhage
   Water and electrolyte imbalance
   Burns
2. *Post-renal:*
   - *Intrarenal:* Sulphonamide crystalluria; and precipitation of uric-acid crystals following treatment with nitrogen mustard, etc.
   - *Extrarenal:* Ureteric and urethral obstruction.

causes[13] have been recorded and Table II summarizes these.

Glomerulonephritis may cause acute oliguric renal failure. As a rule, however, the history of a preceding streptococcal infection in a young subject, and the association of facial oedema and hypertension with proteinuria and haematuria suggest the diagnosis. It should be realized that, while an elevation of systolic blood pressure is common, diastolic hypertension and retinopathy are not usual features of acute renal failure from tubular necrosis. Hypertension may be present with gross sodium and water overload, and has been seen most frequently in post-traumatic cases. Furthermore, oedema usually occurs only if overhydration is present or as a result of the underlying state, e.g. congestive heart failure. Periorbital oedema, except in cases following carbon tetrachloride and chloroform intoxication,[8] is not a usual feature of the condition.

With underlying chronic renal disease, it may be difficult to differentiate a superimposed acute episode from acute renal failure *de novo*. Although the history may be helpful, radiological evaluation of the size of the kidneys is mandatory. The small kidneys of chronic renal disease may be demonstrated — this is in contrast to the normal-sized or slightly enlarged kidneys of acute tubular necrosis.

Our cases of acute renal failure have been drawn from the 3 main racial groups of the Cape Peninsula. There were 24 Coloured, 17 White and 14 Bantu patients. The important clinical details are recorded in Table I. These cases of acute renal failure have been subdivided into 2 groups; 64% in group I and 36% in group II.

Group I includes obstetric and nephrotoxic causes and miscellaneous causes such as infections, intravascular haemolysis and hepatorenal failure of uncertain origin. Group II is limited to postoperative and post-traumatic cases where there is rapid and extensive tissue destruction, often with infection and characterized by rapidly progressive uraemia. This differentiation is important, since the prognosis is very different in the 2 groups.

Apart from the cases attributable to acute tubular necrosis there was 1 patient with suspected peri-ureteric fibrosis in which, however, acute tubular necrosis could not be excluded; 2 with severe acute pyelonephritis, one of whom had proximal tubular necrosis as well; 3 patients with renal cortical necrosis; and 1 with 'failed reflow' kidney.[14] In assessing the mortality of acute renal failure the last 2 conditions mentioned are frequently excluded on the grounds that they are irreversible. This is not justifiable, since these conditions occur in the same clinical setting and, furthermore, they frequently cannot be distinguished during the oliguric phase. Two patients with non-oliguric acute renal failure are included because this is a variant of acute tubular necrosis.

Clinical Settings

The clinical settings are conveniently grouped as follows: (1) obstetric; (2) medical; (3) postoperative, and (4) post-traumatic.

The majority of the 25 obstetric cases followed infected abortions, many of which, it is believed, were induced. No less than 23 of the 27 patients of Russell *et al.*[15] who had abortions in the first trimester were criminally induced. Apart from mechanical interference, commonly soap and water, concentrated chloroxylenol ('dettol') or unknown corrosives are introduced into the uterus. The relationship between *Cl. welchii* infection and acute renal failure[16] has been stressed. In 5 cases (cases 7, 12, 13, 17 and 21) *Cl. welchii* infection was proved. In 4 further cases (cases 11, 19, 20 and 22) it was strongly suspected on clinical grounds. In some of these, intravascular haemolysis was severe.

Nephrotoxic cases numbered 2 only, but in 2 other cases (37 and 48) kanamycin was a likely contributory cause. Of the miscellaneous group of 8 cases, 3 unusual instances should be noted, viz. cerebral malaria (without haemoglobinuria), protocoproporphyria, and fulminating beriberi. In contradistinction to several other reports, mis-matched transfusions do not feature in this series. In only 2 cases was that possibility considered, and in only 1 could it not be excluded. The postoperative cases include several with major arterial surgery, and 2 cardiac cases in which hypothermia was employed. The traumatic cases resulted from 6 motor accidents and 1 train accident. The injuries were severe and multiple, involving fractures of long bones, crushing of the thorax and multiple visceral injuries. Hypotension was the major precipitating factor in most of these cases.

REVERSIBILITY OF THE RENAL LESION

Acute renal failure from tubular necrosis is reversible. The truly irreversible conditions are: extensive bilateral renal cortical necrosis, rare cases of glomerulonephritis and of severe pyelonephritis with renal medullary necrosis, and vascular obstruction to both kidneys. Renal cortical necrosis may be suspected from the history — it usually follows concealed

accidental haemorrhage, but other causes have been recognized.[17] Two of our cases were post-traumatic (cases 50 and 52), and one followed acute fulminating beriberi (case 35). Complete anuria, macroscopic haematuria, especially in the first few days of severe oliguria, and prolongation of profound oliguria beyond 3 weeks, are features favouring the diagnosis of bilateral cortical necrosis. In cases of doubt and when anuria continues past the 3rd week, renal biopsy may help, but this may be hazardous because of the bleeding tendency of the uraemic state. We have, therefore, been reluctant to use this procedure. Furthermore, biopsy need not necessarily assist in assessing the extent of the necrosis. Although the study of routine biopsy samples is of considerable academic interest, it is of little or no practical assistance.

Apart from renal cortical necrosis, complete anuria may also occur in acute diffuse glomerulonephritis and in a post-renal obstructive lesion — the most frequent cause of complete anuria. In post-renal obstructive lesions the clinical features, cystoscopy, ureteric catheterization and retrograde pyelography usually establish the diagnosis, but peri-ureteric fibrosis is a source of difficulty. Ormond[18] has recently emphasized the importance of severe low backache, and abdominal and flank pain, which often precede the onset of oliguria or, more usually, anuria. The picture is further complicated by symptoms of uraemia and of secondary infection. Pyuria was present in 24 of 34 patients.[19] The presence of oliguria or anuria with no recognizable cause of obstruction is suggestive, especially when urine flow is re-established following ureteric catheterization. The importance of intravenous urography in demonstrating widening of the upper ureter and pelvis, in contrast to narrowing of the mid-ureter with medial deviation, has been stressed.[16]

COURSE AND MANAGEMENT

The course and management demand consideration together since mismanagement can materially alter the course and produce disastrous results. In order to gauge the course during the oliguric and early diuretic phases, daily estimation of the concentrations of blood urea or serum creatinine and serum electrolytes is essential, in addition to meticulous measurement of fluid intake and output and regular weighing, where possible. The daily urine volume and the blood-urea or serum-creatinine estimations provide the main yardsticks in assessing progress.

The mean duration of the oliguria in 35 'recovered' patients was 11·1 days (±6·5) and varied between 4 and 29 days. Findings similar to these have been reported by others.[20,21] While the division into early and late diuretic phases has been defined as the point at which the first sustained downward trend in the blood-urea level occurs,[21] we have preferred to note the '1-litre day'. The mean duration for the period, onset to '1-litre day', was 14·6 days (±7·4) and varied between 5 and 37 days. In general, with the start of the diuretic phase, a stepwise increase in urine output occurred until the '1-litre day' was reached 4 - 5 days later. Unlike the findings 10 years ago, when basic fluid intake was 750 - 1,000 ml. per 24 hours, the urine volumes rarely exceeded 3 litres in the diuretic phase.

With the onset of oliguria the blood-urea and serum-creatinine concentrations rose rapidly. At the same time the fall in serum bicarbonate was profound, frequently to levels in the vicinity of 10 mEq./l. Hpyocalcaemia occurred concurrently with a marked rise in serum inorganic phosphate, often to levels of 10 mg. per 100 ml. or more. Hyperkalaemia (potassium — 6 mEq./l. and above) occurred in 28 patients (> 7 mEq./l. in 11, and > 8 mEq./l. in 4 patients). Where sufficient data were available, the mean daily rise in blood urea during the oliguric phase was calculated. In group I the rise in both dialysed and undialysed patients was 35 mg. per 100 ml. In group II the rise was considerably greater — 48 mg. per 100 ml. The clinical course of our recovered patients in group I has, in general, resembled that described by Loughridge *et al.*[21] — despite the frequency of severe infection in the obstetric cases.

Notwithstanding wide publicity concerning the importance of a restricted fluid intake, 6 patients (5 of whom died) were overhydrated when first seen by us (cases 3, 9, 24, 36, 48, and 53).

The management of acute renal failure has been well reviewed,[22] but in order to assess our results it is necessary to consider our practice, including the use of haemodialysis.

Careful explanation of the management to the patient is important. Initially the patient may feel perfectly well and resent the restrictions of food and fluid imposed on him. Most of our patients were drawn from the underprivileged section of the community. The intelligent, well-informed patient usually has little difficulty in understanding the purpose of the restrictions, but considerable tact and patience are necessary to overcome the ignorance and fear of some patients. Explanation to these patients in their own language through an interpreter, where necessary, is essential.

We have avoided the use of indwelling urethral catheters, except in patients with local complicating pathology. Only if necessary is the patient catheterized initially to obtain a urine specimen for diagnosis. Early mobilization of bed patients (and physiotherapy for those confined to bed), and isolation with barrier nursing is important in preventing infection — a prime cause of death in acute renal failure.

The 'Conservative' Régime

Restriction of the fluid intake and the use of an electrolyte-free carbohydrate solution is the basis of therapy.

Following admission to hospital, assessment of the state of water and electrolyte balance, although often difficult, must be made before the maintenance programme is instituted — overhydration is likely to be present. After judicious correction of any major losses, daily maintenance fluids are set at 400 - 500 ml. per day. In the absence of any great variation in the breathing, temperature or humidity, this allows for adjustment to insensible respiration at a rate of 0·5 ml. per kilo. per hour and for water of oxidation on a protein-sparing régime. Additional basic fluids to cover the losses during fever [+ 200 ml. (?) for every degree over 100°F.] or the very hot weather of the local summer are difficult to estimate, and we have exceptionally set the intake at 750 or even 1,000 ml. per 24 hours. A record of the *daily weight* provides the best guide. With appropriate fluid intake the patient should lose 250 - 500 G. per day.

Replacement fluids consist of an amount equal to the previous day's urine output plus any gastro-intestinal aspirate or loss from vomiting and/or diarrhoea. Owing to the poor gastro-intestinal tolerance of these patients, we have abandoned the use of oral fat emulsions as a means of decreasing protein metabolism. Nor do we believe tube feeding offers any advantage. An amount of 100 G. glu-

cose a day provides a near-maximal protein-sparing effect.[23] We have found oral lactose more palatable than glucose, and most patients tolerate a 15 - 20% concentration well with minimal diarrhoea. Where intravenous administration is necessary, we have avoided indwelling venous cannulae because of the very real danger, particularly in uraemic patients, of suppurative thrombophlebitis following infection with a resistant staphylococcus. We have abandoned the intracaval use of concentrated glucose (40%) in view of the great hazard of thrombosis.[10] A 10 - 15% invert-sugar or glucose solution, sometimes combined with ethyl alcohol, is used. The alcohol provides 6 calories per ml. and should not be infused faster than 15 ml. per hour. The rate of infusion of the glucose and alcohol mixture can be calculated from this basic information.

During the oliguric phase the use of glucose or lactose only is often essential, but in practice many patients with normal or slightly raised serum potassium are given the following: small helpings of apple charlotte, ice cream, custard, maize-flour puddings and potassium-free carbonated sweet drinks — ginger beer has been a great standby. Glucose sweets are allowed *ad lib*. Small slivers of crisp toast with occasional cups of black tea add variety and help to maintain the morale of many patients. Any bread and butter used is salt-free, and fruit juices and high-potassium foods are forbidden. Allowance is made for the fluid content of the food. When, however, nausea or vomiting occurs, the intravenous route is used — preferably with a fine needle in a peripheral vein.

Electrolytes are withheld during the oliguric phase except for replacement of gross losses. Gastric and intestinal losses are conveniently replaced by 0·5-N saline or fluid containing an anionic pattern similar to that of plasma with regard to choride and bicarbonate ions. Like some, we usually replace urine volume by water only, but others add electrolytes, depending on the urinary losses. Usually no attempt is made to correct hyponatraemia, since excessive amounts of cation need to be infused. Only occasionally is correction of acidosis with M/6 sodium lactate necessary. Calcium gluconate may be used to combat hypocalcaemia. Appropriate electrolyte replacement in the diuretic phase is required when sodium and potassium losses are considerable, but no good purpose is served by forcing electrolytes at this stage, since the restoration of tubular function is usually rapid. During the diuretic phase there is a rapid fall in blood urea, and once levels of about 150 mg. per 100 ml. are reached at 20 G. protein diet is instituted.

Hyperkalaemia must be viewed in relation to the course of acute renal failure. In addition to the daily (or twice daily) estimation of potassium, ECG monitoring for evidence of hyperkalaemia is necessary. Hypocalcaemia and hyponatraemia potentiate the effect of a high serum potassium and should be corrected when gross. In group II cases, and when there is much infection with rapid tissue destruction, the serum potassium may rise sharply, out of proportion to the other biochemical indices, and is often at dangerous levels before the end of the first week. These levels may be reached rapidly and serum-potassium concentration per litre may rise more than a milli-equivalent in the course of 24 - 48 hours. In such cases the rise is countered by the administration of glucose and insulin (1 G. glucose per unit of insulin). It is unnecessary to use cation-exchange resins prophylactically in all cases, but they are of value where the serum-potassium level has shown a sharp upward trend. The oral administration of a sodium-cycle resin* in amounts of 35 - 45 G. per day in ginger beer has been effective. A continuous infusion of a standard mixture of calcium and sodium with glucose may also be effective,[24] but we are reluctant to use it as part of our daily régime, despite the claim that sodium administration has no dire effects.

If these measures fail, dialysis should be performed without delay. If uraemic symptoms are present in addition to hyperkalaemia, the measures for reducing serum potassium outlined are used temporarily before urgent dialysis. We have observed that in 3 out of 5 post-abortal patients with marked intravascular haemolysis from severe *Cl. welchii* infection, the serum potassium failed to rise with the advance of the other clinical and biochemical indices of acute renal failure.

Other Measures

McCracken and Parsons' claim[25] that an anabolic agent, norenthandrolone, ('nilevar') was capable of reducing protein catabolism by approximately 70% in obstetric cases, but not in post-traumatic cases, needs to be confirmed. Norandrostenolone ('durabolin'), 25 mg. by injection every second day, has been given for 7 - 15 days in 21 cases, but we have been less impressed with its value. It should be noted that the obstetric cases in which it is claimed to be effective are, in any event, those with the best prognosis.

Antibiotics have not been used prophylactically, although they have not been withheld in cases of infection. Due regard is given to the disappearance rates of the appropriate antibiotic in serum. Penicillin, erythromycin, chlortetracycline, 'active' 'chloromycetin', and 'active' novobiocin behave normally.[26,27] In uraemic patients the glucuronic-acid conjugates of chloromycetin persist for many hours in the serum and their potential toxicity is unknown. When infection is already present, as it is in many cases, these antibiotics may be used with a normal loading and slightly reduced or normal maintenance dosage, with the possible exception of chloromycetin. Whenever possible the use of streptomycin, the other tetracycline antibiotics, and the nephrotoxic drugs, neomycin and kanamycin, has been avoided, since their half-lives are considerably prolonged. Polymyxin B and vancomycin tend to accumulate in uraemia and must be used with the utmost caution.

In addition to penicillin treatment, antigas-gangrene serum has been administered in all cases of suspected *Cl. welchii* infection. Where vomiting and restlessness are troublesome, promazine, chlorpromazine and trifluoperazine have been used in low dosage. A transfusion of fresh packed cells has been given when the haemoglobin level has fallen below 8 G. per 100 ml.

Haemodialysis

While many patients can be satisfactorily managed on the above régime, in some haemodialysis is necessary. The indications have been reviewed elsewhere,[28] but in general

* CTS-3 carboxylic-acid-type resin (sodium salt) supplied by courtesy of Eli Lilly & Co.

more weight is given to the early clinical signs and symptoms as opposed to the abnormal serum chemistry (marked hyperkalaemia excepted). Although it is frequently difficult to differentiate the milder symptoms of uraemia from those of severe infection, our practice is to dialyse even when symptoms are mild. Of the 55 patients, 30 underwent dialysis, and 6 needed a further dialysis. Of these patients, 16 were from the obstetric, 1 from the nephrotoxic, 4 from the miscellaneous, 5 from the postoperative, and 4 from the post-traumatic group.

Dialysis is carried out in a room specially designed for the purpose. The procedure is described in the appendix. Apart from occasional sudden hypotension at the start of dialysis, no great variation in the blood pressure has been observed during the procedure. As the dialysis proceeds the blood pressure may tend to rise, but the use of hypotensive agents has been unnecessary. The temperature has usually remained unchanged, and haemolysis has not occurred.

Sedation may be necessary to allay restlessness. Pethidine or an intermediate-acting barbiturate is usually given. Unless the patient is already getting an antibiotic, penicillin, ½ mega unit, is given twice in the 24 hours.

Not only may the drugs given be dialysable, but the electrolyte concentration may influence their action. In a digitalized patient a sudden drop in serum potassium may precipitate digitalis intoxication, with the development of dangerous arrhythmia. Regular ECG control is therefore essential throughout the procedure.

Apart from the bursting of defective coils on 2 occasions, intermittent irregularity of the arterial inflow in 4 patients, and intermittent venous obstruction in 1, the procedure has been trouble-free and accomplished without fatality. Deaths during dialysis have, however, been recorded.[21] The main complication has been transient hypotension. Other complications have included a case of ventricular tachycardia in 1 patient, troublesome epistaxis in 2 and gastro-intestinal haemorrhage in 2. In 1 the haemorrhage was of such gravity that it led us to abandon dialysis after 3 hours. In 4, cut-down wounds became infected.

Biochemical Changes caused by Dialysis

The improvements following dialysis are shown in Table III. The reduction in the blood-urea and serum-creatinine levels is considerable. Hyperkalaemia is corrected in all patients and acidosis relieved or greatly ameliorated in most. Abnormal sodium and chloride concentrations show a return towards normal levels. The phosphate and uric-acid concentrations are reduced materially, while hypocalcaemia, if present, is corrected. While all cases show biochemical improvement, it is clear that without dialysis certain patients (e.g. case 19, Fig. 1) would not have survived.

TABLE III. MEAN AND STANDARD DEVIATIONS OF BLOOD AND SERUM VALUES

Substance	*Before dialysis*	*After dialysis*
Blood urea—mg. per 100 ml. (31)	376 ±111·6	182 ± 82·3
Serum creatinine—mg. per 100 ml. (23)	15·4± 4·8	8·6± 3·1
Serum potassium — mEq./l. (30)	6·6± 1·6	4·6± 1·0
Serum CO_2—mEq./l. (24) ..	10·9± 4·2	19·3± 4·9
Serum sodium—mEq./l. (23)	133·7± 4·4	135·9± 3·6
Serum chloride—mEq./l. (25)	88·8± 7·6	93·6± 6·0

Bracketed figures represent number of observations.

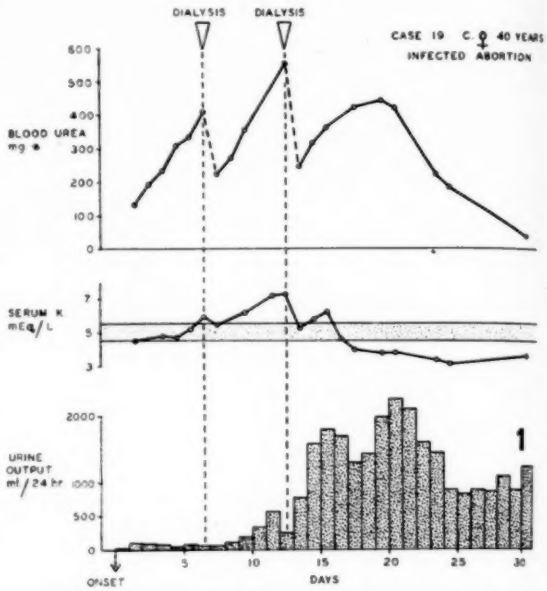

Fig. 1. Illustrating severe uraemia necessitating dialysis on 2 occasions.

The magnitude of the fall in the blood-urea and serum-creatinine levels was considerable, averaging 186 and 6·7 mg. per 100 ml. respectively. The fall in blood urea exceeded 300 mg. per 100 ml. in only 2 patients, and 200 mg. per 100 ml. in 11; it was less than 100 mg. per 100 ml. in 3. In the earlier cases bath water was not changed.

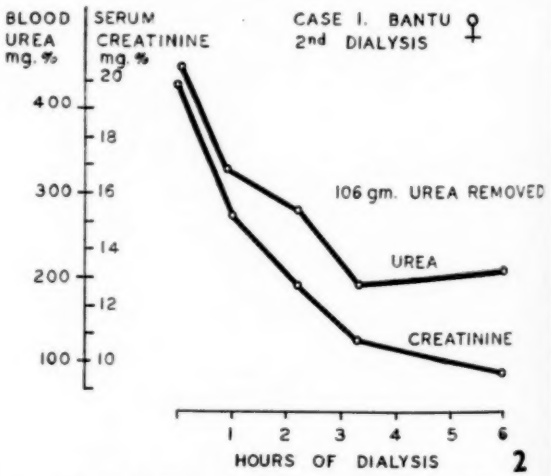

Fig. 2 Unlike urea, creatinine continues to fall.

and hence the fall was less than in subsequent cases. In addition, however, there are marked differences in the clearance of urea at comparable levels of blood urea. Variation in dialysance of urea may be related to the tightness of the coil packing, resulting in inefficient perfusion, and the recently suggested remedies[32] have been adopted.

Changing the bath fluid influenced the fall in blood-urea concentration, but not that of creatinine (Fig. 2). There is a rapid fall in blood-urea concentration in all cases up to 3 hours and a levelling out thereafter in cases in which bath fluid is unchanged. When the fluid is changed the level continues to fall. The serum-creatinine level falls gradually throughout dialysis and changing the bath water has little effect.

Fig. 3 compares the urea levels in serum and bath fluid. With the fall in urea concentration there occurs a

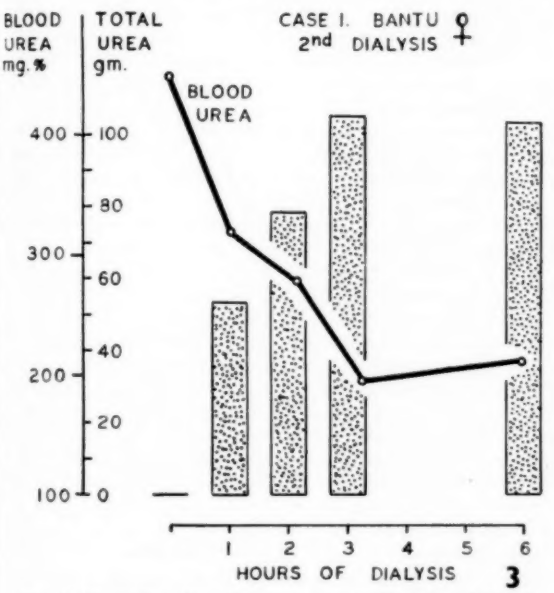

Fig. 3. Illustrating the changes in concentrations of blood and bath urea during dialysis without change of bath water. (Shaded areas represent bath-urea concentrations.)

reciprocal rise in the bath urea — to as much as 139 G. Potassium behaves similarly. Normal serum-potassium values are achieved by the end of the second hour. Although the increase in bath potassium is small when the initial bath concentration is 5 mEq./l., it has amounted to 350 mEq. with a bath concentration of 2·5 mEq./l. Gjørup and Thaysen, when dialysing against a potassium-free solution throughout, found the amount of potassium removed to average 419 mEq.[33] Although this exceeded the calculated excess potassium in their patients, the serum values remained within normal limits.

Bacteriological and chemical investigations have provided no explanation for the alteration in the colour of the bath fluid, which changes gradually to a yellow-green colour. This is maximal at the end of 1 - 2 hours in some cases. A characteristic odour is also present.

RESULTS

Of 25 patients managed conservatively, 16 survived. Of the 9 who died, 7 were considered to have died as a result of the primary condition or its complications. One of these died of septicaemia when well over the acute renal failure. Case 28 died as a result of fulminating enterocolitis, while case 25 died from uraemia before dialysis could be started. Thirty patients were dialysed with 15 deaths, but there were no fatalities during dialysis.

While dramatic improvement frequently followed haemodialysis, the final assessment is judged simply as death

TABLE IV. RESULTS OF TREATMENT IN 55 CASES OF ACUTE RENAL FAILURE ††

	Patients	*Dialysed**	*Died*	*% Mortality*
Group I:				
Obstetric ..	25	16	5**	20
Nephrotoxic ..	2	1	0	0
Miscellaneous ..	8	4	3†	37
Total ..	35	21	8	23
Group II:				
Postoperative ..	13	5	7	54
Post-traumatic ..	7	4	6	86
Total ..	20	9	13	65

* Six patients were dialysed twice.

** In 3 of the obstetric cases the renal lesion was irreversible—2 with bilateral cortical necrosis and 1 with 'failed reflow' kidney.

† One patient with beriberi had bilateral cortical necrosis.

or survival. The results in all cases are summarized in Table IV, and the causes of death are given in Table V.

In group I, 8 of 35 patients died (23%). Whereas the mortality in the obstetric group was 20%, it should be noted that only 3 of the 5 deaths could reasonably be attributed to uraemia *per se*. Thus, case 24 had an irreversible parenchymal renal lesion ('failed reflow' kidney). Case 9 was *in extremis* with gross overhydration when referred for treatment. Pelvic peritonitis was also present. In case 25, in addition to a liver lesion of eclampsia, there was evidence of commencing recovery from acute tubular necrosis. Autopsy, however, failed to reveal any explanation for her sudden death. In the remaining 2, infection was responsible for the fatal outcome. In case 10 the severity of the septicaemia with acute endocarditis was sufficient in itself to cause death. Case 15 had recovered from the acute renal failure and the blood-urea level had been within normal limits for 12 days, when a recurrence of septicaemia resulted in death on the 22nd day. In the miscellaneous group, 2 of 3 deaths were attributable to uraemia. In the patient with burns (aged 67 years) fluid loss from fulminating diarrhoea contributed materially, while case 35 had bilateral renal cortical necrosis. The third patient died from the primary condition — a mesenteric thrombosis with infarction of the bowel.

In group II, 13 of the 20 patients died (65%). In the

†† Since the submission of this paper for publication, a further 13 patients have been treated, with the following results:

	Patients	*Dialysed*	*Died*
Obstetric	6	3	0
Miscellaneous ..	4	4	2
Postoperative ..	3	2	2

TABLE V. CAUSES OF DEATH

Group	Case no.	Cause of death
Group I:		
Obstetric	9	Septicaemia; uraemia and overhydration
	10	Septicaemia with acute endocarditis
	15	Staphylococcal septicaemia
	24	Uraemia—'failed reflow' kidney
	25	Uncertain—eclampsia
Medical	28	Uraemia—electrolyte imbalance
	30	Mesenteric thrombosis
	35	Uraemia—renal cortical necrosis
Group II:		
Postoperative	36	Staphylococcal pyaemia with pulmonary abscess
	39	Cerebrovascular accident
	41	Pulmonary and popliteal embolism
	42	Cerebrovascular accident and myocardial infarction
	45	Cardiac arrest—myocardial infarction
	47	Pulmonary embolism
	48	Suppurative cholangitis
Post-traumatic	49	Intercurrent influenza and bronchopneumonia
	50	Uraemia—renal cortical necrosis
	51	Uraemia—gastro-intestinal haemorrhage
	52	Uraemia—renal cortical necrosis
	53	Bronchopneumonia and overhydration
	54	Fat embolism

postoperative group there was a 50% mortality rate. In 5 of 7 cases death was attributable to a complication of the primary condition which accompanied acute renal failure and which may have, in any event, resulted in death. Case 45, who died of cardiac arrest, was found at autopsy to have a recent myocardial infarction. In the 7th case there was gross overhydration and severe complicating pyaemia.

Only 1 of the patients in the post-traumatic group survived. Two patients who died had extensive renal cortical necrosis and another had fat embolism. Case 49, with a severe chest injury and multiple fractures of long bones, developed influenza during an outbreak in the ward, while making good progress, and succumbed rapidly from bronchopneumonia. Case 51 died, during the early diuretic phase following the 2nd dialysis, from uncontrollable gastro-intestinal haemorrhage resulting from uraemic ulceration of the bowel. Case 53, who was referred on his fourth oliguric day with gross overhydration, succumbed 2 days later from severe bronchopneumonia. Thus, in this group 3 had irrecoverable disease, 2 died of complicating infection, and 1 of severe haemorrhage ascribable to the uraemic state itself.

DISCUSSION

The necessity for prompt diagnosis of acute renal failure has been emphasized. While there are difficulties in diagnosis, these can be avoided by directing attention to the quantity of urine and its composition. While, in general, blood-urea levels appear to reflect the clinical status of the uraemic patient, discrepancies are well known. Some of our patients were relatively symptom-free with blood-urea levels of over 400 mg. per 100 ml. Hyperkalaemia, acidosis, electrolyte imbalance and uraemic symptoms are integral features of acute renal failure, all of which can be corrected with efficient treatment. This treatment can best be achieved by referring suspected cases *as soon as possible* to a renal centre with an experienced haemodialysis unit. Such centres, with their trained and experienced personnel, are best equipped to deal with the complicated problems of fluid and electrolyte balance, the efficient prevention and control of infection and the other complications of the uraemic state. Early transfer of patients will permit of timely dialysis, and patients should be referred not later than the 3rd day of oliguria. The indications for dialysis have been considered elsewhere.[28] The majority of patients have a good clinical and biochemical result from dialysis, but there are some cases in which the degree of reduction in the blood urea is disappointing. It is our impression, however, that there is a beneficial effect despite very moderate reduction in the blood-urea level.

Death in acute renal failure results from: (1) the underlying primary condition, and (2) acute renal failure and its complications. Apart from an irrecoverable primary condition and an irreversible renal lesion (cortical necrosis), death from acute renal failure should be reduced to a minimum with modern methods of management. In the past the chief causes of death were hyperkalaemia, pulmonary oedema and uraemic coma. In this series there were only 6 recoverable cases in which death was ascribable to uraemia itself. In 2 instances patients were referred when moribund and in an overhydrated state, and in 1 of these infection was also present.

The frequency of the complications of acute renal failure increases with delay in the use of the artificial kidney. The main factors contributing to fatality are:

(*i*) *Infection*. This is the most important factor and has been stressed by many authors.[13,21,24,25] It accounts for from 45% to 72% of the deaths. It should be noted that, while Balch *et al.*[25] pointed out that infection was responsible for 45% of deaths from post-traumatic renal insufficiency, Balch[26] claimed that there was no evidence that patients with post-traumatic renal insufficiency are more susceptible to infection. He considered factors such as extent of tissue damage, delay in treatment, and inadequacy of debridement of wounds, to be of major importance. Nevertheless, infection is frequently present in cases of acute renal failure, and it is difficult to control. In the uraemic state there is delayed wound healing[27] with resultant poor localization of infection. Apart from the possible lethal effect of infection, morbidity is prolonged. Patients should be barrier-nursed under strict anti-infective precautions, and antibiotics should not be used prophylactically. Staphylococcal enteritis has been a much feared complication.[21] Avoidance of unnecessary urethral catheterization, active physiotherapy (especially breathing exercises), and early mobilization all contribute to the prevention of infection.

(*ii*) *Cardio-pulmonary complications*. These include arrhythmias (usually in those with previous cardiovascular disease and/or during digitalis therapy), pulmonary oedema and acute respiratory failure. Pulmonary oedema results from overhydration and should no longer be a frequent and lethal complication of acute renal failure. Nevertheless, at least 15 of Salisbury's 31 fatal cases had

TABLE VI. RESULTS OF TREATMENT OF ACUTE RENAL FAILURE FROM VARIOUS CENTRES

	Group I (obstetric and medical)				Group II (postoperative and post-traumatic)			
	Total	*Dialysed*	*Died*	% *Mortality*	*Total*	*Dialysed*	*Died*	% *Mortality*
Swann and Merrill (1953) [5]	63	17	25	40	22	41	14	64
Anthonisen *et al.* (1956) [39]	17	17	10	59*	18	18	14	78
Bull *et al.* (1956 - 8) [40-43]	67	5	26	39	—	—	—	—
Teschan *et al.* (1958) [6]	—	—	—	—	51	31	27	53
Parsons and McCracken (1959) [31]	38	26	6	16	25	19	18	72
Bluemle *et al.* (1959) [24]	62	29	22	35	38	22	28	74
Hammersmith (1960) [21,44] **	36	17	8	22	74	39	59	80
Jackson *et al.* (1960) [45]	33	18	18	55*	22	17	11	50
Kiley *et al.* (1960) [46]	23	14	2	9	57	35	37	65
Groote Schuur Hospital (present series)	35	21	8	23	20	9	13	65

* The unusually high mortality is attributable presumably to the exclusion of undialysed cases (Anthonisen *et al.*) and to the inclusion of 11 fatal cases of glomerulonephritis (Jackson *et al.*).
** Group I figures are taken from Loughridge *et al.*,[21] and Group II figures from Shackman *et al.*[44].

pulmonary oedema.[20] Most renal units have had to treat patients with overhydration. Despite repeated emphasis, the importance of a restricted fluid and salt intake is not yet sufficiently appreciated.

(*iii*) *Central-nervous-system complications*. Coma and convulsions and the attendant danger of inhalation pneumonia may result in death, and are likely to be seen in advanced cases when referred too late. Almost two thirds of Bluemle's[24] patients with coma developed pneumonia.

(*iv*) *Bleeding*. Numerous bleeding defects have been demonstrated — vascular abnormalities, platelet defects, prothrombin depression, etc., while epistaxis, and oozing from wounds and into injection sites were found in a few patients — in only 1 of our patients was intractable bleeding considered responsible for death.

Comparison of mortality rates from different centres presents difficulties on account of the variable nature of the clinical material and the differences in management. Nevertheless, the mortality rate in this series, when compared with others, reveals a remarkable consistency (Table VI). In general, it is obvious that the mortality in the group I patients (despite the frequency and severity of infection in our cases) was very much lower than that in group II. There is little doubt concerning the gravity of the post-traumatic cases. The mortality from different centres has consistently been above 50% and is often above 70%. The differences may be related to:

1. The severe accelerated uraemia of the postoperative and, in particular, post-traumatic cases. Whereas the daily rate of rise in blood-urea concentration is usually below 35 mg. per 100 ml. in group I patients, it is considerably greater in the group II patients and may amount to 50[6] and 51[10] mg. per 100 ml. (blood-urea nitrogen) for post-traumatic cases.

2. The grave and often fatal nature of the underlying primary condition and its complications.

3. In the postoperative group the greater age and infirmity of many of these patients is undoubtedly a factor which is not sufficiently stressed. In fact, Bluemle *et al.*[24] stated there is no correlation between mortality and age. Kiley *et al.*,[46] in noting the effect of age, also emphasized the bad prognosis in patients following aneurysmectomy.

Of 50 patients with acute renal failure resulting from surgical conditions, 12 were jaundiced, but in no instance did Shackman *et al.*[44] consider jaundice primarily responsible for the condition. Jaundice, from hepatic disease, and acute renal failure have long been held to be a particularly unfavourable combination. Jackson *et al.*[45] have suggested separation of such jaundiced patients within the group of acute recoverable renal failure 'to avoid presenting an unnecessarily poor prognosis'. While none will deny that jaundice may contribute to an unfavourable outcome, the claim that the poor prognosis in all cases of renal failure with jaundice from hepatic disease is primarily attributable to the hepatic disease, is open to question. In their series, no less than 5 of 7 such fatal cases had undergone major surgery and, therefore, we would have included them in any event in group II with its poor prognosis.

In 2 cases in the post-traumatic group the renal lesions were irreversible. It is common to find such cases excluded from a consideration of acute renal failure, but irreversibility may only become apparent after protracted treatment, and there is always a possibility that cortical necrosis may be partial or mild, with a definite, although slight, prospect of survival. The possibility of improving mortality in the potentially reversible cases lies in earlier reference of such cases. Thus, with the avoidance of ill-considered overhydration, the prompt institution of scrupulous anti-infective precautions, and the opportunity for earlier dialysis, some improvement in prognosis may be expected. In the severe cases dialysis should be undertaken well before the 6th day, and probably before the 4th day.

The benefits of prophylactic dialysis have been advocated by Teschan *et al.*[6] and it remains to be seen whether this procedure will materially affect the poor outlook of the post-traumatic cases. The stated aim of maintaining the blood non-protein nitrogen at about 150 mg. per 100 ml., so that patients can enjoy a liberal food intake, has merit, but one can only view with considerable apprehension the prolonged retention of indwelling cannulae, and the statement that transient bacteraemia with fever and chills occurred in some of their patients within an hour of the beginning of dialysis is hardly reassuring. Despite general improvement and 'well-being', marked anaemia and some mental defect persisted, and there were 3 deaths among their 15 patients. There is a case for early dialysis and more frequent repetition, if necessary, but the advantages of daily dialysis have to be established, as has the harmlessness of such a procedure.

While the Kolff-Brigham rotating-drum kidney has been

preferred on the grounds of greater efficiency, there is the possible danger of overhydration, with pulmonary oedema. We have little doubt that the 'fixed' capacity and the advantage of ultrafiltration of the twin-coil kidney more than compensates for its lesser efficiency.

Improvements in the performance of the twin-coil kidney have resulted from minor modifications in design, and the effects of using a coil of longer surface area remain to be explored.[32] It is hoped that unnecessarily frequent dialysis may be avoided.

No matter what procedure is used, the most important means of reducing mortality in patients with severe accelerated uraemia is the avoidance of ill-considered parenteral administration of salt and water, and the prompt transfer to a renal centre. Only then will the distressing experience of death within hours of arrival[31] be avoided.

SUMMARY

Two years' experience in the diagnosis and management of acute renal failure, including the use of the twin-coil artificial kidney, is described.

The cases are considered in 2 groups: group I (medical and obstetric cases) has a very much better prognosis than group II (postoperative and post-traumatic cases).

The factors underlying the differences in mortality are discussed. The avoidance of ill-considered parenteral therapy and the importance of prompt transfer of such patients to a renal centre is stressed.

APPENDIX

Dialysis Procedure

In the event of dialysis, 6 pints of fresh blood are reserved and compatibility tests are carried out against the patient's blood and between each unit of blood as well. The patient is weighed before dialysis and again after the procedure. This is the most accurate way of assessing fluid loss during dialysis (the disposable-coil artificial kidney is an ultrafilter as well as a dialyser). Following skin preparation in the ward, and observing scrupulous aseptic technique, artery and vein exposures are carried out in the artificial-kidney room by surgical colleagues in the artificial-kidney team. Brachial block performed by an anaesthetist has been used in most of our cases with excellent results. The procedure provides very satisfactory anaesthesia and vasodilation. One patient developed a pneumothorax as a direct result of the brachial block, but this complication is rare in good hands. A severe bleeding tendency and any significant respiratory pathology are contraindications to the performance of brachial block. After calibrating the flow rate, the coils are filled to a pressure of 180 mm.Hg and usually need about 1,000 ml. of heparinized blood.

In most of our patients the radial artery (coil inflow) and deep cubital veins (coil outflow) of the same arm have been cannulated. When this has not been feasible, a catheter has been inserted into the inferior vena cava *via* the femoral vein. For venous cannulation we have used an ordinary Baxter infusion set, previously sterilized in 'zephiran'. For the radial artery, a No. 4 polythene catheter has usually been passed with little difficulty, except in very small vessels, when a No. 3 has been used. Smaller calibre catheters do not give adequate flow rates, and in these cases it is better to use the inferior vena cava as the inflow. Once the catheters are *in situ*, they are kept patent by continuous flushing through of heparinized saline.

Heparinization follows — initially 100 units per kilo., and then 1,000 units hourly. Administration is stopped an hour or two before the end of the procedure (usually 6 hours). Although the clotting time was initially repeatedly checked with the aim of keeping it between 15 and 20 minutes, in practice it is so markedly prolonged with the above dosage that we now only carry out the procedure in the last hours of dialysis. We have had no experience with regional heparinization.[29,30] Oozing from cut-down sites, especially if covered, should be carefully looked for throughout dialysis. Oozing from sores on the lips has been an irritating, but minor, complication in some patients. Repeated firm packing is usually sufficient to control oozing. Where oozing or other bleeding is severe, protamine sulphate (50 mg. per 5,000 units heparin) is given at the end of dialysis. The electrolyte composition* of the bath fluid is similar to that of plasma. Where hyperkalaemia is present, the amount of added potassium chloride is reduced or omitted altogether. A flow rate between 200 and 300 ml. per minute has been used. As dialysis proceeds, this speed diminishes and may be increased if necessary. The bath fluid is changed every 3 hours, and the procedure usually lasts 6 hours.

Initially, blood pressure, pulse and respiration rate are measured every minute, then at 5-minute intervals, and after 1 hour every 15 minutes. The danger period is that which follows immediately on the commencement of dialysis. A sudden drop in blood pressure may occur, probably from an abrupt diminution in the circulating blood volume, since the tubing of the artificial kidney is distensible. This can be prevented by clamping the coil outflow initially for a few seconds and then releasing it. Sudden flooding of the patient's circulation with at least 2 units of citrated blood may, by depressing the serum calcium, cause a drop in the blood pressure. Sometimes the low blood pressure persists; this is usually easily countered by elevation of the foot of the bed and the administration of 200 - 300 ml. of blood.

* Electrolyte composition:

Component	*Milli-equivalents per litre*					
	Na+	*K+*	*Ca++*	*Mg++*	*Cl−*	*HCO3−*
NaCl ..	97	—	—	—	97	—
$NaHCO_3$ ..	36	—	—	—	—	36
KCl ..	—	5	—	—	5	—
$CaCl_2$ ..	—	—	5	—	5	—
$MgCl_2$ ..	—	—	—	1·5	1·5	—
Total ..	133	5	5	1·5	108·5	36

Invert sugar 0·4%. Lactic acid to adjust pH to 7·4.

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ACUTE COR PULMONALE AS A COMPLICATION OF PNEUMONIA IN CHILDREN

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Chronic cor pulmonale has been observed frequently in children and has been reported most commonly as a complication of mucoviscidosis.[1] It has been described also in the following: tuberculosis, severe kyphoscoliosis, children suffering from primary vascular disease of the lung,[2-5] asthma,[6] and the Hamman-Rich syndrome.[7]

Acute cor pulmonale, on the other hand, has received little recognition as a complication of acute pulmonary conditions in childhood. The only unequivocal cases of acute cor pulmonale in children have been described by Royce,[1] viz. 2 fatalities caused by calcium chloride crystals in infused plasma. Controversial viewpoints are expressed on whether or not cardiac failure occurs as a complication of acute pneumonia. Royce[1] had seen a few babies, suffering from acute bronchiolitis, who had developed cardiac failure and who had recovered on digitalis therapy.

In an epidemic of acute bronchiolitis, Heycock and Noble[8] noted that the more severe cases, especially those with a fatal outcome, had shown signs of congestive cardiac failure. Similarly, Simpson,[9] in reviewing the case histories of 50 infants with heart failure, found 6 children in whom failure had been secondary to acute pulmonary infection. The Editor of the 'Year book of pediatrics',[10] who commented on this paper, doubted that heart failure was a complication of bronchiolitis and suggested that basal emphysema might be responsible for the engorged neck veins, the enlarged liver and the basal râles. Disney *et al.*,[11] commenting on an epidemic of bronchiolitis in infants, did not consider cardiac failure as a cause of death in their cases. The old concept of 'toxic' myocarditis complicating pneumonia appears to have been discarded.[12]

At Baragwanath Hospital we not infrequently encounter acute cor pulmonale as a complication of pneumonia in childhood. It is our purpose to describe the clinical features, the outcome and the treatment of this condition.

MATERIAL AND METHODS

This investigation is based on 178 consecutive Bantu children below the age of 9 years, who were admitted to 2 wards of the Baragwanath Hospital with pneumonia during the winter of 1958. X-ray examination of the chest was performed in all cases and electrocardiograms (ECGs) were obtained in 124 patients.

There were 155 children with bronchopneumonia. In the remaining 23 patients the bronchopneumonia was associated with pertussis. One child presenting with an acute pneumonia was later proved on lung biopsy to be suffering from the Hamman-Rich syndrome, but has been included in this series because of the acute mode of onset.

The patients were carefully examined for evidence of cor pulmonale; this was diagnosed in the presence of the following combination of signs: central cyanosis; increased venous pressure as shown by engorged neck veins and by enlargement of the liver; a hyperdynamic circulation characterized by a large pulse pressure, capillary pulsation and warm, moist extremities; clinical evidence of right ventricular dominance shown by a forceful left parasternal thrust; and the presence of a gallop rhythm. The ECGs were analysed for evidence of right ventricular hypertrophy and/or peaked P waves greater than 2·5 mm. in height.[13-15] Cardiomegaly was considered to be present on the X-ray film if the cardiothoracic ratio was greater than 55%.

Penicillin was the principal antibiotic used in the treatment of these children; usually 250,000 units were given every 6 hours by intramuscular injection. The antibiotic was changed if pyrexia had not subsided and the clinical condition of the patient had not improved within 72 hours of the commencement of therapy. If there was severe dyspnoea, cyanosis and restlessness, the child was placed in an oxygen tent.

Digoxin was given to 11 patients. The digitalizing dose was calculated on the basis of 0·08 mg. per kg. body weight, given in 3 equally divided doses by mouth at 8-hourly intervals. The maintenance dose consisted of $\frac{1}{4}$ - $\frac{1}{8}$ of the total digitalizing dose, and was given once daily. The drug was discontinued as soon as the signs of cardiac failure had disappeared.

RESULTS

There were 14 children who fulfilled the criteria for the diagnosis of cor pulmonale. Their ages ranged from 5 months to 4 years. One of the patients was 5 months old, 7 were aged 12 - 24 months and 6 were 25 - 48 months old.

Of the 14 children, 12 suffered from bronchopneumonia (4 cases being complications of pertussis), 1 suffered from giant-cell pneumonia, and the remaining 1 from the Hamman-Rich syndrome. Of the 4 children with pertussis bronchopneumonia, 1 had been whooping for 5 months, 2 for 2 months, and 1 for 3 days.

Central cyanosis was a well-marked feature in 12 of the 14 patients. In the remaining 2 children cyanosis was not easily recognizable because the children were anaemic. Their respective haemoglobin values were 8·5 and 8·8 G. per 100 ml. In contrast, only 14 (8%) of the remaining 164 patients with pneumonia were clinically cyanosed.

All the children with cor pulmonale had hyperdynamic circulations when first seen, as compared with 45 (27%) of the patients with uncomplicated pneumonia.

A presystolic gallop rhythm was found on auscultation in all 14 children. This finding was confirmed in 2 patients in whom phonocardiography was carried out. The disappearance of the gallop rhythm was the first sign of improvement in the 12 patients who recovered. The other 2 children died within 36 hours of admission to hospital, and the presystolic gallop rhythm persisted up to the time of death.

The 14 children with cor pulmonale were too ill for X-ray examination on admission to hospital, and this investigation was deferred until some improvement had been noted in their condition. Despite this delay, 8 of them showed radiological evidence of cardiomegaly. Furthermore, there was a return to normal heart size within 7 - 14 days in those patients who recovered.

No ECG abnormalities were detected in the patients with uncomplicated pneumonia. In the group with cor pulmonale, 8 (57%) showed evidence of right ventricular dominance, and of these 4 had enlarged, peaked P waves, i.e. P 'pulmonale'.

All 14 children received antibiotics and oxygen therapy, while 11 were given digoxin. On this therapy the gallop rhythm and the signs of cardiac failure disappeared within 24 hours in 5 patients and after 48 hours in a further 5. In 1 patient, who developed an empyema, signs of heart failure lasted for 4 days, and the patient suffering from the Hamman-Rich syndrome recovered from cardiac failure 7 days after admission to hospital.

There were 2 deaths. At autopsy, one child, aged 3 years, showed evidence of a giant-cell pneumonia and excess fat in the liver. The heart showed right ventricular hypertrophy and weighed 75 G. as compared with the average normal of 58 G. in this age group. The other child, aged $1\frac{1}{2}$ years, had a severe necrotizing pneumonia. The heart, which showed evidence of right ventricular hypertrophy, weighed 67 G. as compared with an average normal weight of 52 G. at this age. There was no histological evidence of myocarditis in either of these children.

DISCUSSION

Our investigation shows that cor pulmonale is a not uncommon complication of severe acute pulmonary infections among children admitted to this hospital. This series does not give a true indication of the incidence of cor pulmonale, since many children suffering from pneumonia or whooping cough who attend at this hospital are treated as outpatients, and only the more seriously ill gain admission to the wards. Thus, an incidence of cor pulmonale of 7·8% in patients suffering from acute lung pathology refers only to the more seriously affected children.

The finding of a presystolic gallop rhythm in every patient with cor pulmonale requires comment. The additional heart sound is heard best just internal to the apical cardiac impulse and towards the left sternal border in the 4th intercostal space. It is an audible atrial sound.[10,11] So constant was this finding that we have included it among the diagnostic criteria for cor pulmonale. Furthermore, we found this sign to be of prognostic value, since its disappearance was followed by rapid clinical improvement.

It was of particular interest in 2 children. They were markedly dyspnoeic and cyanosed, with signs of heart failure and a presystolic gallop rhythm. They were given antibiotics and placed in oxygen tents. On examination the following day the signs of cardiac failure and the gallop rhythm had disappeared. When they were taken out of the oxygen tent, cyanosis reappeared and the gallop rhythm returned. With renewed administration of oxygen both the cyanosis and the gallop rhythm disappeared again. This phenomenon was experienced several times during the acute phase of the disease. These observations lend weight to the suggestion that anoxia (hypoxia) is the basis for the pulmonary hypertension, the increased cardiac output, and the cardiac failure in cor pulmonale.

The ECG and the X-ray examinations were of limited value in the diagnosis, since they showed right ventricular strain or cardiomegaly, respectively, in only half the patients.

Of the 23 patients with whooping-cough bronchopneumonia, 4 developed the clinical features of acute cor pulmonale. It would appear that the heart failure supervened in pertussis only when it was complicated by bronchopneumonia for, during the period under review, many patients with pertussis, unaccompanied by pneumonia, were seen in the outpatient department and none showed evidence of cor pulmonale.

Additional evidence that we were dealing with cor pulmonale was supplied by the autopsies performed on the 2 fatal cases. In both instances there was cardiac enlargement with right ventricular hypertrophy in association with pneumonia.

In the belief that hypoxia was the prime factor in the aetiology of our cases, we decided to omit digoxin in the last 3 patients and treat them with oxygen and antibiotics only. In these, and in 7 subsequent patients not included in this series, recovery from acute cor pulmonale

occurred within 48 hours of commencement of therapy. Thus, the prompt administration of oxygen is the single most important therapeutic measure in cases of acute cor pulmonale, even in the absence of clinical cyanosis, which may be masked by an underlying anaemia. From this experience it is suggested that digoxin be withheld unless signs of heart failure persist 48 hours after antibiotic and oxygen therapy is begun.

SUMMARY

Cor pulmonale is a well-described complication of chronic pulmonary disease, but is rarely mentioned as a complication of acute pulmonary infection in childhood. This series shows that, of 178 consecutive cases of severe pneumonia, there were 14 complicated by acute cor pulmonale. These included a patient with an acute onset of the Hamman-Rich syndrome.

The symptoms and signs of cor pulmonale in childhood are described, and special emphasis is placed on the development and prognostic significance of the presystolic gallop rhythm.

The clinical diagnosis of cardiopulmonary disease is emphasized as being more important and more accurate than either the ECG or radiological findings.

Autopsy evidence of right ventricular hypertrophy was found in 2 fatal cases of pneumonia and cor pulmonale. Children with pertussis may develop cardiac complications when there is an associated bronchopneumonia.

Hypoxia is the probable basis for the hyperdynamic circulation and the resultant cardiac failure. Prompt administration of oxygen was successful in overcoming right heart failure.

Our thanks are due to Dr. I. Frack, Superintendent of Baragwanath Hospital, for permission to publish. We are also indebted to Dr. E. Kahn, senior paediatrician, for allowing us access to the cases in his wards, as well as for his invaluable help and criticism.

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BILATERAL TUMOUR-FORMING PULMONARY AMYLOIDOSIS

AN UNUSUAL AND INTERESTING CASE

E. ORSMOND, M.R.C.S., L.R.C.P., D.P.H., *Uitenhage*

A European male, aged 83 years, was admitted to the Uitenhage Provincial Hospital on 5 February 1959 suffering from developing gangrene of his left foot, which was found to be cold, discoloured and slightly oedematous.

He was a free patient, and the house surgeon in charge did the usual routine tests; except for a blood pressure of 180/110 mm.Hg and signs of emphysema, nothing was found in his urine, blood, heart, nervous system, and abdomen to suggest any serious disease. His clinical condition was described as fairly good.

On X-ray examination, however, a rare condition was found. The patient was questioned regarding his lungs and he stated emphatically that he did not remember ever having suffered from any complaint of his chest, but his house doctor stated that he had treated the patient some years ago for pleurisy; this was evidently correct because on postmortem examination there was no doubt that at some time during his life he had definitely suffered from pleurisy.

The hospital radiologist showed me the X-ray film and I decided to show it to the doctors in charge of the tuberculosis section. I was advised that whatever happened I had to retain the lungs at death. Arrangements were then made with the next-of-kin for a postmortem examination in the event of the patient's death.

The patient lived for about a year after this and died in Graaff-Reinet. His body was returned to Uitenhage. At the postmortem examination I found both lungs equally affected; hence I removed the right lung only. I also found that he had a pleural effusion and old pleuritic adhesions of both lungs. The heart appeared to be normal. I was informed that death was due to a stroke.

The case proved so interesting to me and others that it was suggested that I follow it up by sending the lung for a pathological report. This is submitted herewith, together with the X-ray report.

PATHOLOGICAL REPORT ON LUNG

Macroscopic Features

The lung shows the presence of chronic pleurisy. The lung substance is studded with multiple, various-sized, calcified irregular masses, and bears a superficial resemblance to a pneumolithiasis. The calcified masses vary from very small ones to masses several centimetres in diameter. They can be readily enucleated. On dissection they are found to be attached to the walls of the pulmonary veins and grow outwards in irregular positions compressing, but not invading, the contiguous lung substance. They have not been found in the lumina of vessels, nor attached to bronchi.

Chemistry

Chemical examination shows the bulk of the masses to consist of a protein matrix impregnated with calcium, magnesium, and sodium salts (carbonates and chlorides). Carbohydrate is present in traces — tests for fat are negative.

Histology

Histological examination shows that the masses are amorphous, irregular, hyaline masses which consist of *paramyloid* in which calcification has occurred. Tests for amyloid show a red metachromasia with gentian violet and a red colouration with Congo red.

Histological examination of several sections shows that the condition begins as an amyloid degeneration of the media of blood vessels, especially veins. Large collections of plasma cells are found in the vicinity of many of the lesions.

Diagnosis

Primary pulmonary tumour-forming amyloidosis.

RADIOLOGICAL REPORT

11 March 1959 — Chest (Fig. 1)

There are multiple irregular densities scattered throughout both lung fields. These opacities are extremely dense and there is evidence of involvement of some of the bronchi and possibly of some of the blood vessels in the lungs. There is surprisingly little distortion of the lung anatomy. These appearances are consistent with a diagnosis of pneumopathia osteoplastica racemosa in which bone formation occurs within the lung tissue and bronchial tube walls. Note also the wedging of the body of one of the lower dorsal vertebrae. This probably represents senile osteoporosis with wedge compression of the involved vertebra.

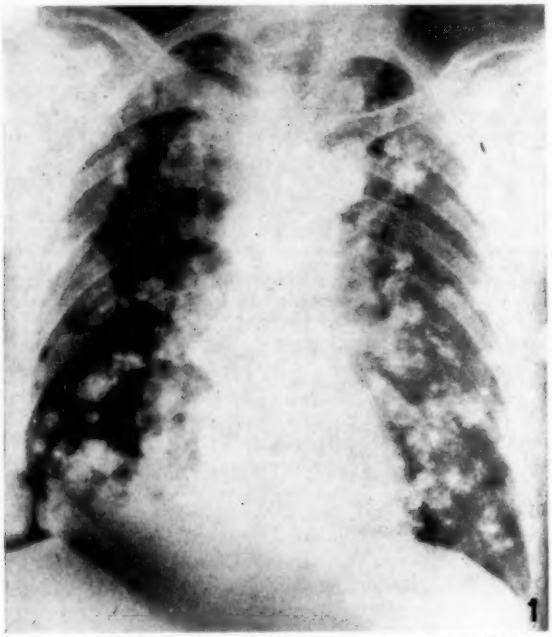

Fig. 1. Chest X-ray showing calcified masses in both lung fields.

DISCUSSION

Frank Bergman and Erland Linder[1] give the following information about amyloidosis:

'Incomplete knowledge of the aetiology and nature of amyloid degeneration makes classification of amyloid diseases difficult. Research has recognized three groups in addition to the secondary amyloidosis of chronic infectious diseases, namely (1) Primary atypical amyloidosis characterized among other things by the absence of any disposing basal disease; (2) nodular solitary or multiple local amyloidosis; and (3) amyloidosis, associated with multiple myeloma amyloid tumours, has been the subject of research for many years, especially in Germany where Leopold emphasized the value of publishing cases of localized amyloidosis and stressed the importance of descriptions of the tumours in the elucidation of problems bearing on amyloidosis.'

The factors considered of importance for the development of secondary amyloidosis can nearly always be excluded in nodular amyloidosis.

Another important difference is that amyloid tumours are seldom seen in parenchymatous organs. They show a liking for smooth muscle, skin, lymph nodes, and the mucous membrane of the digestive and urinary tracts. This form of amyloidosis is rarely seen in the lung tissue. As far as the lung itself is concerned, changes sometimes produce symptoms of pneumonia and pleurisy, but at other times no clinical signs are present and are only discovered incidentally by X-ray examination. It will be noticed from the above history that this was what occurred in the present case, when it was only by X-ray examination that the strange condition was discovered. This form of amyloidosis is rarely seen in the lung parenchyma, and a search in the literature failed to reveal reports of more than 13 cases.

My thanks are due to Prof. B. J. P. Bekker of the University of Pretoria for the pathological report, and to Dr. T. B. Forrest of Bloemfontein for the radiological report. Without their cooperation it would have been impossible for me to publish this case.

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THE PRACTICE OF MEDICINE IN THE COMMONWEALTH OF AUSTRALIA

C. S. JONES, M.B., CH.B., *Department of Anaesthesia, Prince Henry Hospital, and The University of New South Wales Medical School, Sydney*

As in the United States, each of the 6 States forming the Commonwealth of Australia has its own Medical Board which is responsible for maintaining a register of medical practitioners and acts as a licensing body. These Boards do not have any disciplinary powers (beyond withdrawing the names of practitioners who do not comply with the ordinary requirements for registration such as the payment of an annual fee), the conduct of members of the profession being subject to the ordinary rules of society as interpreted by the courts of law and to the weight of professional opinion as represented by the British Medical Association. For purposes of registration the Australian Capital Territory of Canberra is included in New South Wales and the Northern Territory in South Australia.

The Honorary System

Medical practice follows the same lines as elsewhere in the British Commonwealth, but the honorary system still prevails, particularly in New South Wales. Unlike America, only a small part of the population (about 40%) are private patients either in their own right or by virtue of their regular contributions to insurance schemes. The balance are public patients entitled to free hospital and medical treatment in the many hospitals established by the various states. In these hospitals the public patients are cared for by the 1st and 2nd year residents (Intern and Senior Intern), supervised by Registrars and Honoraries. In consequence the hospitals are inefficient and the public patients inevitably receive care of a rather lower standard than their richer brethren. There is evidence that this last outpost of the Honorary system (an admirable practice originally, but now hopelessly out-dated by modern medical needs), is slowly crumbling, and in centres such as Melbourne an increasing number of full-time and part-time specialist posts are being provided. It is common knowledge that the standard of medical practice is higher in Melbourne than elsewhere in the Commonwealth.

There appears to be a trend towards group practice, among both specialists and general practitioners. Where no specialist register is kept, such groups may be heterogeneous, and it is not uncommon for general practitioners to carry on a specialist consultant practice at another address in addition to their own general practice.

Because the honorary system is not conducive to the most

TABLE I. REQUIREMENTS OF THE VARIOUS STATES

State	*Population (30 Sept. 1958)*	*Number of practitioners registered (May 1961)*	*Number of practitioners resident (May 1961)*	*Specialist register*	*Sout African degrees registrable*	*Remarks*
New South Wales ..	3,769,000	6,067	4,388	No	UCT Wits. Pretoria Natal	The Republic of South Africa is regarded as part of the British Commonwealth (as from 1 June 1961) for purposes of registration
Victoria	2,771,000	5,590	4,014	No	UCT Wits. Pretoria Natal	Must appear in person before Medical Board
Queensland	1,425,000	1,616	1,467	Yes (422)	None	Must satisfy professors of professional knowledge and ability and prove 3 years' practice in medicine
South Australia ..	927,000	1,681	1,181	No	UCT Wits. Pretoria Natal	'The medical degrees of the South African Universities are still acceptable'. Must interview President or member of Board
Western Australia ..	714,000	1,200	900	Yes (173)	None	Must make personal application at Board office
Tasmania	347,000	341	341	No	UCT Wits. Pretoria Natal	Applicant for registration must be a British subject

TABLE II. CLASSIFICATION OF DOCTORS BY CATEGORIES AND STATES

State	*General list*		*Specialists*	*Salaried doctors*	RMOs	*Total*
	Metropolitan	*Country*				
N.S.W. and A.C.T.	1,556	895	1,099	191	705	4,446
Victoria	1,190	498	781	118	511	3,098
Queensland	293	371	313	64	294	1,335
South Australia and Northern Territory	342	190	250	63	140	985
Western Australia	255	134	187	46	142	764
Tasmania	75	56	88	15	63	297
New Guinea	93	—	—	—	—	93
Total	3,804	2,144	2,718	497	1,855	11,018

TABLE III. CLASSIFICATION OF SPECIALISTS BY CATEGORIES AND STATES
TOTAL OF 2,718

	N.S.W.	*Vic.*	*Q'land*	*S.A.*	*W.A.*	*Tas.*	*Total*
Ophthalmology	103	57	28	17	16	6	227
Dermatology	53	29	8	10	8	2	110
E.N.T.	54	32	18	10	12	6	132
Surgery	190	175	66	47	29	19	526
Pathology	80	38	13	13	11	5	160
Medicine	138	124	43	48	23	14	390
Paediatrics	32	16	20	11	5	3	87
Orthopaedics	39	14	18	4	10	3	88
Anaesthetics	56	66	17	16	11	4	170
Allergy	13	14	2	3	2	—	34
Gynaecology	104	61	33	30	25	15	268
Psychology }	53	50	17	10	10	3	143
Psychiatry }							
Arthritis	5	1	—	—	—	—	6
Rheumatism	7	2	—	2	2	—	13
Heart	14	3	—	4	—	1	22
Neurosurgery	10	3	2	2	—	—	17
Tuberculosis and chest	20	15	1	5	8	4	53
Neurology	8	8	2	2	4	—	24
Sterility	1	1	—	—	—	—	2
Urology	32	18	11	2	2	—	65
X-ray	87	54	14	14	9	3	181
Total	1,099	781	313	250	187	88	2,718

efficient utilization of hospital beds, there seem to be more hospitals than might be strictly necessary in the more settled parts of the country, but of course, in the sparsely populated areas the problems of providing a medical service are more difficult to surmount than in South Africa and have led to the formation of the Flying Doctor Service.

Training

There are at present 5 medical schools preparing undergraduate students for medical degrees: those of Sydney, Melbourne, Adelaide, Queensland and Western Australia. The latter graduated its first M.B., B.S. students in 1957. The University of New South Wales and Monash University are developing medical schools at present. The total number of doctors graduating *per annum* is about 450, which appears to be grossly inadequate for the population of 10 million, particularly since it is the intention to increase this population as rapidly as may conveniently be possible by natural means and by immigration. The requirements of the various States with particular regard to South African trained medical practitioners are set out in Table I. for those who may be interested, and the distribution of doctors, including specialists, is shown in Tables II and III. These latter tables are provided by Messrs. Permindex Mailing Ltd., and are not completely accurate since not all States have a specialist register. Some specialists have no higher degree and will not be reflected in Table III.

The information set out in Table I. has been obtained directly from the various licensing boards and is accurate to 30 May 1961.

Remuneration of Practitioners

Judging by the various schedules of fees (Workers' Compensation — Commonwealth Benefit, etc.) the remuneration of medical practitioners throughout Australia is somewhat lower than in South Africa and living costs, particularly the rental or purchase of accommodation, is higher, so that the general practitioner must turn his hand to a considerable volume of clinical material in order to make a good living. This fact, combined with the higher ration of public cases, probably accounts for the part-time consultant practice carried on by many general practitioners in those states where no specialist register exists, as well as for the low ratio of anaesthetists to surgeons (1 to 8).

It will be noted (Table II) that, as in South Africa, there are far more practitioners in the large centres than in the country towns, despite the fact that the latter are well provided with hospital accommodation which is continually being expanded and improved. The road and rail systems are being improved steadily throughout the sub-continent and excellent air services exist at very reasonable rates. The first-class fare from Perth to Sydney, a 7½-hour flight by turbo-prop planes, is R88. The distance is close to 2,000 miles.

At present Australia appears to be in a similar position to that of the United States at the turn of the century, all types of industry being developed and new sources of mineral wealth being exploited almost daily. Along with the rest of the world there is a shortage of medical practitioners which cannot be made good from Commonwealth Medical Schools and it is probable that, particularly if more efficient hospital services are developed, this country will be able to absorb a considerable number of practitioners from elsewhere in the near future.

MINES BENEFIT SOCIETY PRACTICE

SOME ASPECTS OF ITS ECONOMICS

A. M. GOLDBERG, M.B., CH.B., *Brakpan*

In the Brakpan - Springs area the panel doctors of the Mines Benefit Society (MBS) have additional sources of income from appointments and from private practice, including medical aid society practice. It is difficult, if not impossible, to calculate what proportion each benefit society contributes to the total costs of running the practice. If any practice were to consist solely of MBS panel patients it would be a simple matter to assess the cost to the doctor for each member of the panel. No such practice exists, but from practical experience and after discussion with many colleagues in the area I feel that such a practice could be constructed in theory, and valuable deductions could result.

An attempt will be made to answer the following questions:

1. What should be an average day's work of a doctor in solus practice?
2. What remuneration should he reasonably expect for this amount of work?
3. What size of panel will keep a doctor fully occupied?
4. What are the expenses of running such a practice?
5. What panel fee should he receive?

1. A Day's Work in the Brakpan - Springs Area

Consulting sessions: 2 hours, both morning and afternoon. The usual sessions are from 10.30 a.m. to 12.30 p.m. and from 4 p.m. to 6 p.m. Although some patients will require 20 - 30 minutes, there are others who can be dealt with in 4 - 5 minutes (e.g. repeat attendances, certificates, or requests for simple prescriptions for headache tablets, etc.). A considerable proportion of each session is occupied on the telephone in answering queries from patients, or in discussions with specialists. I find that on an average I can see about 6 patients per hour, i.e. a total of 24 patients during the consulting sessions.

Hospital rounds: 1½ hours per day, including operations.
Calls: 3½ hours, about 10 calls (limits 5 - 20).
After hours: 4 calls (limits 1 - 8).
Week-ends and holidays: 6 calls per day (limits 3 - 20).

Injuries received on duty are also treated during the day (see para. 5 below).

The usual 'working' day is 8 a.m. — 6 p.m., allowing 1 hour for lunch. Calls made after 6 p.m. are considered to be 'after hours'.

2. Suitable Remuneration

A useful comparison is the new post of 'principal' medical officer in many Transvaal hospitals. The salary is R4,800 *per annum* for a suitably experienced general practitioner. (In this area practically every panel doctor is widely experienced, having been in practice for many years.) The usual period of duty is 7 hours per day and there may or may not be an occasional night or week-end duty. There are sick leave and travel privileges, 50 days annual leave (including public holidays), a holiday bonus of R120 *p.a.*, in some cases quarters and board at low rates, rail concessions and a pension scheme.

With this post as a yardstick the hard-working panel doctor perpetually on call is surely entitled to an income 50% above that of the hospital medical officer, after making provision for some of the latter's privileges? Thus it is submitted that his net income should be R600 per month, and allowance for the privileges mentioned will be made under the heading of expenses in para. 4.

3. Size of Panel

If the MBS patient is to receive full attention from his doctor in his own house, in the doctor's consulting room, and in hospital, I do not consider that the panel doctor can undertake to care for more than 600 families (1,500 individuals). When I commenced practice in this area 16 years ago, the practitioners were able to cope with far bigger panels than this, but the practice of medicine appears to have changed for the better, and doctors are now devoting far more time to each patient in the examination (including special examinations), in record-taking, and in the follow-up. With the increasing complexity of medicine far more discussions than previously are being held with specialists, and much time is

wasted in obtaining specialists' consent for items omitted from the pathetically inadequate and outdated schedule of drugs. I shall refrain from commenting further on the frustrations experienced — it is difficult to assess compensation in terms of money!

4. Monthly Expenses

Rooms	R80-00
Receptionist	100-00
Electricity	16-00
Telephones (including residence)	20-00
Stationery, etc.	6-00
Car (1,500 miles)	60-00
Allowance for receptionist's leave and bonus	10-00
Share of office boy	16-00
Leave allowance (R440 *p.a.*)	36-00
Pension allowance (see para. 2)	50-00
Insurance (public liability, sickness and accident, fire and burglary)	20-00
Depreciation of furniture, equipment, etc. (R1,500 at 10% *p.a.*)	12-00
Interest on capital (R1,500 at 7%)	9-00
'Emergency' telephone service	4-00
Miscellaneous	12-00
	R451-00

5. Panel Fee

It was postulated in paragraph 2 that the panel doctor should receive for his services a monthly income of R600. As the expenses are R451, the total panel fees should be R1,051. Since the panel has 600 members the panel fee per member will be R1-75.

However, the Rand Mutual Insurance Company pays a fee of 12½ cents for each member in employment on a gold mine or ancillary organization, under the provisions of the Workmen's Compensation Act; most families have a member in such employment (exceptions are widow members and continuation members). Since the doctor's working hours, as outlined in paragraph 1, make provision for the treatment of work injuries, the fee paid by the Rand Mutual (less 2½c to make allowance for families without insured workers) should be deducted from the fee paid by the MBS.

Thus the panel fee to the doctor is to be allocated as follows:

MBS	R1-65
Rand Mutual	R0-10
Total	R1-75

There may be additional income from midwifery and anaesthetic fees. However, this will entail extra work not allowed for in paragraph 1 (operations on the doctor's own patients are allowed for, but not procedures on behalf of colleagues). Thus the additional work involved will cut into his leisure time. Moreover, since he is not compelled to undertake this additional work, it is not considered that any such (doubtful) additional income merits consideration.

6. Conclusions

The present panel fee paid by the MBS is R0-70. Sixteen years ago it was 4/6 (+ 10d. in lieu of anaesthetic and assistance fees) and since R2 is now worth only about one-third of the 1945 £1, the panel fee should accordingly be 3 x 4/6 = R1-35. As pointed out in paragraph 3, the panel doctor can now cope with fewer patients than previously and hence the panel fee of R1-65 as derived in paragraph 5 is consistent with this alternative approach to the problem.

This theoretical discussion can be applied to existing mixed practices. As an example (where a doctor devotes 1/3 of his time to a panel of 200 members):

Expenses for 200 members = 1/3 of R451 (para. 4) = R150
Income from MBS and Rand Mutual =
(200 x R0-70) + (200 x R0-05) = R150

It is evident that it is uneconomic for panel doctors, whether they have large or small panels, to accept the present membership fee of R0-70 per family. A case has been presented for the raising of the fee to R1-65.

ABSTRACT

IN VITRO ASSESSMENT OF COMMERCIAL CONTRACEPTIVE JELLIES AND CREAMS*

Fifty-four commercial contraceptive preparations have been studied for sperm-immobilization capacity by 3 different laboratory methods. In 2 of these methods, the highest dilution of the preparation which immobilized spermatozoa in human semen within 20 seconds after thorough mixing has been determined, by manual mixing with an equal quantity of semen in one method, and in the other by electromagnetic mixing with one-fifth volume of semen. In the third method, the greatest number of volumes of semen has been found in which the spermatozoa were immobilized within 20 seconds by one volume of undiluted contraceptive. A wide range of variation in apparent activity was found by the 3 methods between the most active and the least active materials.

There is a partial positive correlation in the ranking results of the contraceptive materials by the 3 mixing methods, when measured by Spearman's rank correlation coefficient. No study has yet been made which demonstrates a positive correlation between laboratory tests of spermicidal activity and contraceptive effectiveness in clinical use for a series of contraceptive jellies or creams. To state categorically that any cream or jelly is clinically superior to any other awaits such an investigation.

* MacLeod, J., Sobrero, A. J., and Inglis, W. (1961): J. Amer. Med. Assoc., **176**, 427.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during September 1961 are gratefully acknowledged:

Met dank word die volgende skenkings gedurende die maand September erken:

Votive cards in Memory of: Geloftekaarte ter Nagedagtenis aan:

Mr. L. Berman *by* Dr. P. W. J. Keet; Mr. E. H. Victor *by* Dr. and Mrs. R. Theron; Dr. H. W. Dyke *by* Dr. and Mrs. A. W. Sichel, Drs. R. and E. Theron, Mrs. M. H. Wacker, Mrs. C. Bligh-Wall, Border Branch (M.A.S.A.), and Dr. and Mrs. J. McCabe; Mrs. N. Smit *by* Dr. H. Levine; Dr. M. Ginsburg *by* Dr. H. Levine.

Total Received from Votive Cards: Totaal Ontvang van Geloftekaarte: R31.30

Services Rendered to: Dienste Gelewer aan:

Dr. C. Freed's late father *by* Dr. L. Hirsowitz and Dr. V. Gorvy.

Mrs. E. Linton *by* Mr. Carl T. Möller.

Dr. R. L. H. Townsend *by* Dr. R. Richards.

Dr. J. W. S. Millar and family *by* Drs. M. Jackson, A. J. van Amerongen, J. Gluckman, F. Stakes and Mr. J. Wolfowitz.

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G. Drummond, R. Coller and A. Hooper.
Roslyn, daughter of Dr. M. Myers, *by* Dr. H. Forer and Dr. L. Shubitz.
Dr. M. Lipworth *by* Dr. L. Knox.
Dr. G. P. de Kock *by* Mr. M. Stein.
Sister M. E. de Kock *by* Dr. P. Lynch.
Dr. and Mrs. H. Lowenstein *by* Dr. M. J. Levin.

Total Received from Services Rendered:
Totaal Ontvang van Dienste Gelewer: R143.55

Donations: Skenkings:

Drs. Harries, Kuschke, Hofmeyr, D. J. Pudifin, and J. S. M. Sililo.

Total Received from Donations:
Totaal Ontvang van Skenkings: R4.20

Grand Total: Groot Totaal: R179.05

PASSING EVENTS : IN DIE VERBYGAAN

Somerset Hospital, Monthly Clinical Evening, will be held on Tuesday 21 November at 8.15 p.m. in the Nurses' Main Lecture Hall, Somerset Hospital, Green Point. All those interested are invited to attend this meeting.

* * *

Mr. Sidney Sacks, orthopaedic surgeon, of Johannesburg, has been invited to open the discussion on 'Anterior fusion of the spine' at the Meeting of the American Academy of Orthopaedic Surgeons to be held in Chicago in January 1962.

* * *

Mothers' Milk Bank. The Johannesburg Mothers' Milk Bank is now in full operation from its office at 803 Ingram's Buildings, Twist Street, Johannesburg, and ample supplies of breast milk are available on prescription at all times. A qualified nurse has been engaged to supervise donors and to ensure hygienic measures in the collection of milk, and strict laboratory control has also been introduced. The successful launching of this project marks a big step forward in the care of babies in South Africa.

* * *

Moedersmelkbank. Die Moedersmelkbank van Johannesburg is nou in werking vanaf hulle kantoor te Ingramsgebou 803, Twiststraat, Johannesburg. Voldoende voorrade is ten alle tye verkrygbaar op voorskrif. 'n Opgeleide verpleegster is aangestel om toesig te hou oor skenkers en om higiëniese insamelingsmaatreëls te verseker. Streng laboratorium kontrole word ook toegepas. Die geslaagde instelling van hierdie onderneming is 'n groot stap vorentoe in kindersorg in Suid-Afrika.

A Postgraduate Course for General Practitioners will be held at the Medical School of the University of the Witwatersrand from Monday 15 January to Saturday 20 January 1962.

Organized by the University's Faculty of Medicine and the Medical Graduates Association, the course will consist of practical demonstrations, ward rounds, symposia and panel discussions, and senior members of the hospital and the University staff will take part.

Applications to attend the course should be made to the Secretary, Medical Graduates Association, Medical School, Hospital Hill, Johannesburg.

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Dr. H. Grusin, physician, of Johannesburg, has returned to his practice after a six weeks' tour in Europe.

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The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 27 November 1961 at 5.10 p.m. in the Institute Lecture Theatre. Dr. G. Falkson, of Pretoria University, will speak on 'Chemotherapy of solid neoplasms'.

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Edendale Hospital, Pietermaritzburg. Regular hospital staff meetings are held each week: Every Monday at 4.45 p.m. there is a combined medico-surgical case discussion, held alternatively in the Department of Medicine (6th floor) and in the Department of Surgery (1st floor); and at 9.30 a.m. each Saturday there is a combined ward round in the Department of Medicine. All medical practitioners and students are invited to attend these meetings.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

DECA-DURABOLIN

Organon Laboratories announce the introduction of Deca-Durabolin — a form of Durabolin which is fully active for 3 weeks following injection and which shows still further separation of the anabolic from the androgenic activity — and supply the following information:

Description. Deca-Durabolin is nandrolone decanoate (19 norandrostenolone decanoate). For those patients in whom weekly injections are impracticable or in whom oral anabolic substances are ineffective in the doses tolerated, Deca-Durabolin provides an outstanding alternative.

Indications. Carcinomatosis: The use of the highly potent and long-acting Deca-Durabolin is specially indicated in patients in whom it has not been possible to give large enough doses of other anabolic agents to obtain optimal effects without serious side-effects. Deca-Durabolin with cytotoxic agents: Whyte, Watson and Turner noted that patients receiving testosterone were able to tolerate much greater doses of cytotoxic agents before marrow depression occurred.

In a study of 165 cases of early and late breast cancer treated with a combination of an alkylating agent and an androgen, Whyte, Watson and Turner advise the use of 50 mg. of Deca-Durabolin intramuscularly every 3 weeks.

Because of its sustained action Deca-Durabolin is particularly suited for the treatment of geriatric patients with or without osteoporosis. It is also recommended for postoperative use, acute renal failure, during corticosteroid treatment, and debility states.

Dosage. 25 mg. or 50 mg. every 3 weeks.

Side-effects. Chronic toxicity studies did not reveal any harmful effects and in particular no evidence of any liver damage was shown.

Presentation. Deca-Durabolin is available in two strengths: 25 mg./c.c. and 50 mg./c.c. Both strengths are packed in 1 x 1 c.c. and 3 x 1 c.c.

Further information may be obtained from Keatings Pharmaceuticals Ltd., P.O. Box 256, Johannesburg.

ZARONTIN

Parke, Davis Laboratories, pioneers in anticonvulsant therapy, announce the introduction of Zarontin, a new anticonvulsant primarily useful in *petit mal* epilepsy, and supply the following information:

Description. Zarontin (ethosuximide, Parke, Davis) is a new anticonvulsant of the succinimide group. Zarontin is closely related structurally to the other well-known and established members of the succinimide group — Milontin (phensuximide) and Celontin (methsuximide).

The use of this new therapeutic agent has resulted in a high level of control of attacks of *petit mal* in more than three-quarters of the patients treated.

On the basis of effectiveness against *petit mal* attacks, mildness of side-effects, and holding ability, Zarontin offers more promise than other agents tested in the past 10 years.

Indications. Zarontin is intended mainly for the treatment of patients with *petit mal* epilepsy.

During the clinical investigation with Zarontin, the results of therapy were studied in more than 400 patients. Improve-

ment was observed in some 80%; 75% or more reduction in the attack rate was observed in 62% of all patients, and complete control in 43%.

The type of epileptic seizure, whether pure *petit mal* or mixed *petit mal*, had considerable bearing on the efficacy of therapy — Zarontin was much more effective in pure *petit mal* than in other types of seizures combined with mixed *petit mal*.

Side-effects. The most frequent reported side-effects have been nausea, gastric distress, drowsiness, dizziness and headache, these symptoms usually occurring singly. Of more than 400 patients treated, less than 20% showed side-effects. To appreciate the relative insignificance of this number, it is well to consider the nature of anticonvulsant therapy, in which the dosage is often increased gradually until side-effects are elicited and then decreased to a satisfactory therapeutic level. Clinical symptoms associated with repeated large doses of Zarontin during the clinical investigation programme have all disappeared following decrease in dosage or discontinuation of the drug. Many symptoms occurring shortly after initiation of therapy have disappeared as medication at the initial, or higher than initial, dosage level continued.

Administration and dosage. Zarontin is administered orally. The recommended initial dose for patients under 6 years of age is 1 capsule (0·25 G.) daily; for patients 6 years of age and older, 2 capsules (0·5 G.) daily. The dose thereafter should be individualized according to the patient's response. Dosage should be adjusted by small increments.

Presentation. Zarontin is supplied in bottles of 50 soft gelatin capsules, each capsule containing 250 mg. of ethosuximide.

Further information is obtainable from Parke, Davis Laboratories (Pty.) Ltd., P.O. Box 24, Isando, Tvl.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING OCTOBER 1961

Pan American Association of Otorhinolaryngology and Bronchoesophagology Congress, Venezuela, 25 February - 1 March 1962. Dr. Charles M. Norris, Secretary, 3401 N. Broad Street, Philadelphia 40, Pa.

World Federation of Neurology, Buenos Aires, 4 - 9 December 1961. The Secretary, c/o National Institute of Nervous Diseases and Blindness, Bethesda, Md.

Second Congress of the Asiatic Society of Haematology, Manila, 28 - 29 December 1961. Dr. V. de la Fuente, Secretary, 303 Bernabe, Pasay City, Philippines.

Health and Tuberculosis Conference, Ibadan, Nigeria, 26 - 31 March 1962. The Secretary, c/o Chest and Heart Association, Tavistock House, Tavistock Square, London, W.C.1.

BOOK REVIEWS : BOEKBESPREKINGS

PSYCHOLOGICAL CARE OF THE CHILD IN HOSPITAL

The Psychological Care of the Child in Hospital. By Agatha H. Bowley, Ph.D., F.B.Psy.S. Pp. 47. Illustrated. 45c plus 3c postage. London: E. & S. Livingstone. 1961.

Dr. Agatha Bowley is a well-known educational psychologist in London and has been closely associated with the psychological problems of sick children for a considerable time. She has written widely and wisely on many facets of child care. This little handbook on the child in hospital is a gem and should be in the hands of all who have to do with a sick child in a hospital ward. It should be read primarily by matrons and sisters who, though normally kind, behave towards children under their care in hospital in an unintentionally cruel manner, on the assumption that the type of discipline they exert is in the interests of the child. It is important that doctors should read this book, especially hospital administrators, paediatricians, and those who call themselves family doctors. Parents should read this booklet, and learn why they should be allowed freer access to the hospital wards. There would be a more harmonious relationship between the family and the hospital, if parents, nurses, and doctors all realized fully what could be done to prepare a child for admission to, and treatment in, hospital.

A wide view of the subject is taken, ranging from an explanation of the child's reaction to hospitalization, and the preparation for his reception, to the attitude of the parents and nurse. The need for occupational (or diversional) therapy is stressed as well as the need for the continuation of his education. The author points out that it is the mother that the child wants beside him when he is ill. Nursing a sick child at home, or sharing in his care in hospital, is the most certain way of ensuring a speedy recovery from illness.

This is now so widely recognized in Great Britain, that many hospitals have facilities for mothers to 'room-in', and so take an effective part in the nursing of their own children in hospital. These facilities are, at present, non-existent in South Africa. If we in this country are to be considered as an outpost of Western civilization, it is high time that one of the refinements of this culture be put into practice, through the provision of more facilities for mothers to stay with their children in hospital. Where this is not feasible, not only should freer visiting be allowed, but mothers should actually be encouraged to come and assist actively in the nursing of their children.

B.E.

DISC LESIONS

Diseases of the Intervertebral Disc and its Surrounding Tissues. By Reuben Rabinovitch, B.A., M.Sc., M.D. Pp. xii + 152. Illustrated. R6.80. Oxford: Blackwell Scientific Publications. 1961.

This monograph is a valuable study concerned with the hypothesis that some systemic disease of connective tissue, and of disc cartilage in particular, first alters the cartilage matrix in discs and that symptom-producing trauma is secondary to such alterations.

To prove this hypothesis, an anatomical and histological study of 30 human spines in different age groups were made. The photographs, including some in colour, illustrating various facets of these studies, are of very good quality. The author's setting-out of his findings and his sober reasoning is most impressive. Unlike many monographs of this nature the end of the book was reached all too quickly.

During the 30-year period ending June 1959, 2,303 papers appeared on the subject of the intervertebral disc.

It is difficult not to lose one's bearings on the vast sea of disc literature or to avoid drowning. However, this excellent monograph acts as an effective pair of water-wings.

M.S.

HEALTH IN INDUSTRY

Health in Industry. A Guide for Engineers, Executives and Doctors. By R. C. Browne, M.A., D.M. (Oxon.), M.R.C.P. (London). Pp. vii + 157. Illustrated. R1.80 net. London: Edward Arnold Publishers. 1961.

This important book, which embraces the health aspects of all the fields of industrial activity commonly encountered, is described by the author as a guide for engineers, executives and doctors. Therein lies its considerable value, for it is through the combined efforts of these persons that the highest degree of industrial health may be attained.

The author, who achieves the unusual feat of sustaining the

interest of layman and doctor alike, deals with such subjects as the health of the executive, absence due to sickness, equipment design, industrial accidents, and fatigue, automation, environmental factors and chemical substances affecting the human being. The writing is simple and clear and the approach essentially practical. Despite its small size, the book is a mine of valuable information. Naturally, due to its limited compass, detail must sometimes be sacrificed, but this deficiency is successfully overcome by the listing of recommended reading at the end of each chapter for those who wish to pursue further any particular subject.

This book, which, incidentally, is moderately priced, could with advantage be on the bookshelves (and frequently on the desks) of all whose positions in industry determine the well-being of their fellow workers. There are few doctors, who number individual workers of any rank among their patients, who would not be well rewarded by careful perusal of its contents.

M.G.W.

INTRA-ABDOMINAL CRISES

Intra-abdominal Crises. By Kenneth D. Keele, M.D., F.R.C.P. and Norman M. Matheson, F.R.C.S., M.R.C.P., F.A.C.S. Pp. xii + 397. Illustrated. Durban: Butterworth and Co. 1961.

In this little book the authors have set themselves the task of looking at intra-abdominal crises from a new angle — that of arriving at a diagnosis by the process of exclusion when an emergency, the nature of which has to be determined, arises, either from without or within the abdomen. This approach, they say, is one that has received too little attention when such emergencies have to be dealt with, especially in the earlier stages. Their other point is that it is just during these early stages that one is not always able to classify an intra-abdominal emergency as either medical or surgical, without the early assistance of radiology or biochemistry. These should be made to play a more important rôle with advancing technique.

While I agree with all this, I must point out that besides the fact that these procedures can be not only time-consuming and often not infallible, they would certainly tend to increase the cost of hospitalization and treatment, an important consideration these days to many patients.

No surgical procedures are discussed, but suggestions as to management are freely given in the text and will be found useful. The authors have tried to confine themselves to the diagnosis of intra-abdominal crises in their early stages and the diagnostic data at these stages, so that the book should be useful to all who have to deal with acute cases and their early diagnosis and treatment in or outside a hospital.

The book is divided into 3 parts: 'General Features of Intra-abdominal Crises', in which are discussed the background including the patient, the first observer, G.P., ambulance and the hospital; the next deals with a large number of 'Local Abdominal Diseases' including urological and gynaecological conditions; while the last deals with 'Intra-abdominal Crises as part of General Disease'.

The authors appear to have achieved their purpose well, and their subject matter is well presented. There is a short appendix to which reference on points of biochemistry and haematology could be made. It is a pity that the X-ray photographs are on the whole very disappointing, most of them being too small or too soft to show details well enough. The book should nevertheless be useful to most doctors who usually are the first to see cases of intra-abdominal crises in their earliest stages.

G.C.A.v.d.W.

MEDICINE IN TROPICAL AFRICA

Medicine in Tropical Africa. By Michael Gelfand, *C.B.E.*, M.D., F.R.C.P., D.P.H., D.M.R. Pp. 243. Illustrated. R3.00 net. Edinburgh: E. & S. Livingstone. 1961.

This book could appropriately be called a companion volume to Gelfand's *The Sick African*. Whereas in the latter text-book disease states are dealt with as individual or related conditions according to the conventional pattern, there is still the need for disease in the African to be dealt with on broad lines and along comprehensive concepts. On the other hand, it is fallacious to speak of the African as if uniform rules apply to him as regards liability to disease. Dr. Gelfand points out how different the disease incidence may be in the different social strata (he classifies them into preliterate, semiliterate and literate) as also the different beliefs as regards aetiology of disease among them. It is quite clear that prevailing ideas such as the rarity of peptic ulcer or, in contrast, the high incidence of primary carcinoma of the liver in the African, must be qualified. Peptic ulcer is actually quite common in the Buganda; and, whereas cancer of the liver is common in certain regions, it is rare in others where carcinoma of the oesophagus may be very common .

With the big advances in medicine in recent times, the ordinary text-book can no longer afford the space for leisurely discussions and personal observations which have not reached the stage of proof. We have in this book — to give but two examples — the reasons given (from different sources) why the infant does not get malaria in the first 4 months of life in communities where practically everyone harbours the parasites, and why children under 3 years do not have bilharzia even though exposed to infection. And why after 30 years of age 70% of the adult population (in a community where over 80% are infected), no longer pass the ova of bilharzia in either the stools or the urine.

Dr. Gelfand states that tuberculous pericarditis is common amongst his patients (p. 22), but does not make clear later, when he discusses cardiac cases with, predominantly, ascites, how many of these are pericardial. A great deal has been written on the cirrhosis of the African. The form peculiar to him is certainly portal, but everyone would not agree with the author that it is synonymous with Laennec's.

On page 144 we are probably dealing with optic neuritis and not papilloedema, and the converse on page 212. There is no mention of the reports of Foy and Kondi of the correction of their megalocytic anaemia by penicillin.

The book conveys to a great extent Dr. Gelfand's very considerable experience of medicine in the African. In the best Oslerian tradition he has a wide experience of personally conducted postmortems, so that much of what he has to say is more than conjecture. A final point of interest — it is surprising that there should be so little pyelonephritis considering the high incidence of bilharzia with bladder and ureteral involvement.

F.F.

HARTCHIRURGIE

Prosthetic Valves for Cardiac Surgery. Editor-in-chief: K. Alvin Merendino, M.D. Associate Editors: Andrew G. Morrow, M.D., C. Walton Lillehei, M.D., and William H. Muller, jnr. M.D. Pp. xxix + 586. Illustrated. R6.60. Oxford: Blackwell Scientific Publications. 1961.

Op 9 en 10 September 1960 is daar in Chicago, V.S.A., 'n konferensie gehou wat gegaan het oor kunskleppe vir hartchirurgie. Hierdie boek bevat die voordragte wat by daardie vergadering gelewer is sowel as die besprekings wat plaasgevind het. Nie minder nie as 152 hartchirurge en navorsers van oor die hele V.S.A. het aan hierdie konferensie deelgeneem, en onder hulle is al die toonaangewende hartchirurge. Dit is dus nie verbasend dat hierdie boek 'n geweldige hoeveelheid oorspronklike en insiggewende navorsingswerk bevat nie.

Die boek bestaan uit vier afdelings. Die eerste afdeling gaan oor die fisiologie van klepfunksie en oor hartmetabolisme. In die tweede afdeling word die materiaal vir, die ontwerpe van, en die metodes van toets van kunshartkleppe bespreek. Die derde afdeling gaan oor kunskleppe vir die mitraalklep, en die vierde oor kunskleppe vir die aortaklep. By die deurlees van hierdie boek staan die leser verstom oor die uitgebreide navorsingswerk wat op hierdie gebied in die V.S.A. gedoen word. Tegelykertyd word hy ook beïndruk deur die geweldige probleem van die vraagstuk om 'n geskikte kunshartklep te vind en besef hy hoeveel navorsingswerk nog gedoen moet word voordat 'n ideale kunshartklep beskikbaar sal wees.

Alle chirurge en navorsers op die gebied van oophart-chirurgie sal hierdie boek sekerlik graag wil besit.

B. J. van R. D.